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Logon file405 02jun09 07:11:02
           *** ANNOUNCEMENTS ***
                    ***
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Derwent World Patents Index First View Overview (File 331)

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NEW FILE
***File 457, The Lancet(R)
                    ***

RESUMED UPDATING
***File 523, D&B European Financial Records
                  ***
RELOADS COMPLETED
***File 658, TRADEMARKSCAN(R) - Benelux

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***File 659, TRADEMARKSCAN(R) - Denmark
***File 661, TRADEMARKSCAN(R) - Switzerland
***File 662, TRADEMARKSCAN(R) - Austria
***File 669, TRADEMARKSCAN(R) - Japan
***File 678, TRADEMARKSCAN(R) - Norway
                    ***
FILES REMOVED
***File 301, CHEMNAME - please use File 398 ChemSearch
***File 388, PEDS: Defense Program Summaries
***File 588, DMS-FI Contract Awards

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SYSTEM:HOME
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Menu System II: D2 version 1.8.0 term=ASCII
                     *** DIALOG HOMEBASE(SM) Main Menu ***

 Information:
  1.  Announcements (new files, reloads, etc.)
  2.  Database, Rates, & Command Descriptions
  3.  Help in Choosing Databases for Your Topic
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etc.)
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  6.  DIALOG(R) Document Delivery
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 service.  Enter a BEGIN command plus a file number to search a
database
(e.g., B1 for ERIC).
? b 410

       02jun09 07:11:03 User226352 Session D1140.1
            $0.00    0.275 DialUnits FileHomeBase
     $0.00  Estimated cost FileHomeBase
     $0.00  Estimated cost this search
     $0.00  Estimated total session cost   0.275 DialUnits

File 410:Dialog Customer Newsletters 2008
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      Set  Items  Description
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? set hi ;set hi
HILIGHT set on as ''
HILIGHT set on as ''
? b biochem
       02jun09 07:11:06 User226352 Session D1140.2
            $0.00    0.117 DialUnits File410
     $0.00  Estimated cost File410
     $0.02  TELNET
     $0.02  Estimated cost this search
     $0.02  Estimated total session cost   0.392 DialUnits

SYSTEM:OS  - DIALOG OneSearch
  File   5:Biosis Previews(R) 1926-2009/May W4
         (c) 2009 The Thomson Corporation
  File   6:NTIS 1964-2009/May W4
         (c) 2009 NTIS, Intl Cpyrght All Rights Res
  File  24:CSA Life Sciences Abstracts 1966-2009/Jul
         (c) 2009 CSA.
  File  34:SciSearch(R) Cited Ref Sci 1990-2009/May W4
         (c) 2009 The Thomson Corp
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  File  41:Pollution Abstracts 1966-2009/Jul
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  File  45:EMCare 2009/May W4
         (c) 2009 Elsevier B.V.
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         (c) 2009 CAB International
*File  50: The file has been reloaded and accession numbers
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  File  71:ELSEVIER BIOBASE 1994-2009/May W5
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  File  72:EMBASE 1993-2009/May 29
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  File  73:EMBASE 1974-2009/May 29
         (c) 2009 Elsevier B.V.
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  File  98:General Sci Abs 1984-2009/Jun
         (c) 2009 The HW Wilson Co.
  File 103:Energy SciTec 1974-2009/May B1
         (c) 2009 Contains copyrighted material
*File 103: For access restrictions see Help Restrict.
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  File 136:BioEngineering Abstracts 1966-2007/Jan
         (c) 2007 CSA.
*File 136: This file is closed.
  File 143:Biol. & Agric. Index 1983-2009/May
         (c) 2009 The HW Wilson Co
  File 144:Pascal 1973-2009/May W5
         (c) 2009 INIST/CNRS
  File 154:MEDLINE(R) 1990-2009/May 29
         (c) format only 2009 Dialog
  File 155:MEDLINE(R) 1950-2009/May 29
         (c) format only 2009 Dialog
  File 156:ToxFile 1965-2009/May W4
         (c) format only 2009 Dialog
  File 162:Global Health 1983-2009/May W4
         (c) 2009 CAB International
*File 162: The file has been reloaded and accession numbers have
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         (c) 2009 Elsevier B.V.
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         (c) 2009 Royal Soc Chemistry
*File 305: Alert feature enhanced for multiple files, duplicate
removal, customized scheduling.  See HELP ALERT.
  File 369:New Scientist 1994-2009/May W4
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  File 393:Beilstein Database - Abstracts 2008/Q2
         (c) 2008 Beilstein GmbH
  File 399:CA SEARCH(R) 1967-2009/UD=15023
         (c) 2009 American Chemical Society
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  File 434:SciSearch(R) Cited Ref Sci 1974-1989/Dec
         (c) 2006 The Thomson Corp

      Set  Items  Description
      ---  -----  -----------
? s adiponectin
      S1   49381  ADIPONECTIN
? s s1 and (multimer or aggregat?)
           49381  S1
            9927  MULTIMER
         1297324  AGGREGAT?
      S2     464  S1 AND (MULTIMER OR AGGREGAT?)
? rd s2

>>>Duplicate detection is not supported for File 393.
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>>>Records from unsupported files will be retained in the RD set.
      S3     110  RD S2  (unique items)
? t s3/7/1=10
>>>'=' not allowed in command
? t s3/7/1-10
>>>Format 7 is not valid in file 143

 3/7/1     (Item 1 from file: 5)
DIALOG(R)File   5:Biosis Previews(R)
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0020883139   BIOSIS NO.: 200900223473
Comparison of Immunoassays for the Selective Measurement of Human
  High-Molecular Weight Adiponectin
AUTHOR: Liu Dan; Schuster Tibor; Baumann Marcus; Roos Marcel;
Sollinger
  Daniel; Lutz Jens; Heemann Uwe; von Eynatten Maximilian (Reprint)
AUTHOR ADDRESS: Tech Univ Munich, Dept Nephrol, Ismaningerstr 22,
D-81675
  Munich, Germany**Germany
AUTHOR E-MAIL ADDRESS: maximilian.eynatten@lrz.tum.de
JOURNAL: Clinical Chemistry  55 (3): p568-572 MAR 2009 2009
ISSN: 0009-9147
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: BACKGROUND: Adiponectin is an adipocyte-derived hormone
  circulating in different multimer complexes. The
  high-molecular-weight (HMW) complex is likely the active form of
this
  protein and has been recognized as a risk marker for type 2
diabetes and
  coronary artery disease (CAD). Because quantification of HMW
  adiponectin by Western blot analysis is time-consuming, novel
  ELISAs have been developed to simplify measurements in clinical
research.
  However, these enzyme immunoassays have not been cross-validated in
  larger patient groups. We evaluated 2 individual ELISA systems by
  comparison to Western blotting for measurement of the distribution
of HMW
  adiponectin in healthy individuals and patients with CAD and type 2
  diabetes.METHODS: We measured HMW adiponectin in 204 individuals
  (83 CAD patients, 81 type 2 diabetes patients, and 40 healthy
controls).
  Correlations, range of agreement, and imprecision of HMW
concentrations
  obtained using 2 commercial ELISAs (#1, ALPCO Diagnostics; #2,
Millipore)
  were evaluated by comparison with quantitative Western
blotting.RESULT:
```

Adiponectin results of the ELISAs were significantly correlated with those obtained by Western blotting (both r > 0.75, P < 0.001). Deming regression and Bland-Altman analyses indicated high agreement among the 3 immunoassays. The median difference between HMW adiponectin concentrations measured by ELISA and by Western blot was +0.4 mg/L for ELISA #1 and -0.4 mg/L for ELISA #2 with 95% of value differences < 3 mg/L.CONCLUSIONS: Selective measurement of HMW adiponectin by ELISA is feasible; however, individual differences among immunoassays must be considered. The evaluated ELISAs exhibit analytical characteristics that allow their use as equivalent for Western blot analysis in larger clinical and epidemiological groups.

3/7/2 (Item 2 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

0020786289 BIOSIS NO.: 200900126623
Fish oil, but not flaxseed oil, decreases inflammation and prevents pressure overload-induced cardiac dysfunction
AUTHOR: Duda Monika K; O'Shea Karen M; Tintinu Anselm; Xu Wenhong; Khairallah Ramzi J; Barrows Brian R; Chess David J; Azimzadeh Agnes M; Harris William S; Sharov Victor G; Sabbah Hani N; Stanley William C (Reprint)
AUTHOR ADDRESS: Univ Maryland, Dept Med, Div Cardiol, 20 Penn St,HSF2,Room S022, Baltimore, MD 21201 USA**USA
AUTHOR E-MAIL ADDRESS: wstanley@medicine.umaryland.edu
JOURNAL: Cardiovascular Research 81 (2): p319-327 FEB 1 2009 2009
ITEM IDENTIFIER: doi:10.1093/cvr/cvn310
ISSN: 0008-6363
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: Clinical studies suggest that intake of omega-3 polyunsaturated fatty acids (omega-3 PUFA) may lower the incidence of heart failure. Dietary supplementation with omega-3 PUFA exerts metabolic and anti-inflammatory effects that could prevent left ventricle (LV) pathology; however, it is unclear whether these effects occur at clinically relevant doses and whether there are differences between omega-3 PUFA from fish [eicosapentaenoic acid (EPA) and docosahexaenoic acid (DHA)] and vegetable sources [alpha-linolenic acid (ALA)].We assessed the development of LV remodelling and pathology in rats subjected to aortic banding treated with omega-3 PUFA over a dose range that spanned the intake of humans taking omega-3 PUFA supplements. Rats

were fed a standard food or diets supplemented with EPA+DHA or ALA at
0.7, 2.3, or 7% of energy intake. Without supplementation, aortic banding
increased LV mass and end-systolic and -diastolic volumes. ALA
supplementation had little effect on LV remodelling and dysfunction. In
contrast, EPA+DHA dose-dependently increased EPA and DHA, decreased
arachidonic acid in cardiac membrane phospholipids, and prevented the
increase in LV end-diastolic and -systolic volumes. EPA+DHA resulted in a
dose-dependent increase in the anti-inflammatory adipokine
adiponectin, and there was a strong correlation between the
prevention of LV chamber enlargement and plasma levels of
adiponectin (r = -0.78). Supplementation with EPA+DHA had anti-
aggregatory and anti-inflammatory effects as evidenced by decreases
in urinary thromboxane B-2 and serum tumour necrosis factor-alpha.Dietary
supplementation with omega-3 PUFA derived from fish, but not from
vegetable sources, increased plasma adiponectin, suppressed
inflammation, and prevented cardiac remodelling and dysfunction under
pressure overload conditions.

3/7/3 (Item 3 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

0020723112 BIOSIS NO.: 200900063446
Differential effects of bariatric surgery-induced weight loss on
adiponectin multimer complexes
AUTHOR: Linscheid Philippe (Reprint); Christ-Crain Mirjam; Stoeckli Rolf;
Mueller Beat; Keller Ulrich
AUTHOR ADDRESS: Univ Basel Hosp, Dept Res, CH-4031 Basel, Switzerland**
Switzerland
JOURNAL: International Journal of Obesity 32 (Suppl. 6): pS77 DEC 2008
2008
CONFERENCE/MEETING: 4th Fribourg Obesity Research Conference Fribourg,
SWITZERLAND 20070914,
ISSN: 0307-0565
DOCUMENT TYPE: Meeting; Meeting Poster
RECORD TYPE: Citation
LANGUAGE: English

3/7/4 (Item 4 from file: 5)

0020720253 BIOSIS NO.: 200900060587
High molecular weight adiponectin correlates positively with myeloperoxidase in patients with type 2 diabetes mellitus
AUTHOR: Bobbert P (Reprint); Rauch U; Stratmann B; Goldin-Lang P; Antoniak S; Bobbert T; Schultheiss H P; Tschoepe D
AUTHOR ADDRESS: Charite Univ Med Berlin, Med Clin 2, Dept Cardiol and Pneumol, Campus Benjamin Franklin,Hindenburgdamm 30, D-12203 Berlin, Germany**Germany
AUTHOR E-MAIL ADDRESS: peter.bobbert@charite.de
JOURNAL: Diabetes Research and Clinical Practice 82 (2): p179-184 NOV 2008
2008
ITEM IDENTIFIER: doi:10.1016/j.diabres.2008.07.018
ISSN: 0168-8227
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: Adiponectin (APN) is present in human plasma as a low molecular weight (LMW), a middle molecular weight (MMW) and a high molecular weight form (HMW). As a support to determine properties such as anti-atherogenic or atherogenic effects, recent clinical studies suppose to deter-mine the ratio of each APN multimer to total APN but not the absolute plasma concentration of APN. In the present study, the correlation of APN and its multimers with myeloperoxidase (MPO), an enzyme with pro-inflammatory proper-ties, was examined in patients with type 2 diabetes mellitus.MPO and APN serum levels were assessed in 49 patients with type 2 diabetes mellitus at the beginning and at the end of an anti-diabetic treatment. After treatment a significant increase in the ratio of HMW to total APN (from 0.43 +/- 0.16 to 0.59 +/- 0.14, $p < 0.05$) was found. Before treatment, HMW-APN was correlated positively with MPO ($r = 0.314$, $p < 0.05$). Moreover, a positive correlation was observed between the increased HMW ratio and MPO during treatment ($r = 0.304$, $p < 0.05$).HMW-APN correlates positively with MPO in patients with type 2 diabetes. Therefore, HMW-APN may exert possible pro-inflammatory effects in type 2 diabetes.

3/7/5 (Item 5 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

0020587007 BIOSIS NO.: 200800633946
Adiponectin multimer distribution in patients with familial combined hyperlipidemia
AUTHOR: Koenen Tim B (Reprint); van Tits Lambertus J H; Holewijn Suzanne;
Lemmers Heidi L M; den Heijer Martin; Stalenhoef Anton F H; de Graaf Jacqueline
AUTHOR ADDRESS: Radboud Univ Nijmegen, Med Ctr, Dept Gen Internal Med 463,
POB 9101, NL-6500 HB Nijmegen, Netherlands**Netherlands
AUTHOR E-MAIL ADDRESS: T.Koenen@aig.umcn.nl
JOURNAL: Biochemical and Biophysical Research Communications 376 (1): p
164-168 NOV 7 2008 2008
ITEM IDENTIFIER: doi:10.1016/j.bbrc.2008.08.111
ISSN: 0006-291X
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: Adiponectin is secreted from adipocytes in different multimers, of which the high molecular weight (HMW) form is supposed to
mediate favorable metabolic and anti-atherogenic effects. We determined
adiponectin multimers in 29 female and 22 male patients with familial combined hyperlipidemia (FCH) and 51 age-, gender-, and BMI-matched controls in relation to cardiovascular disease (CVD). We observed a clear sexual dimorphism of total adiponectin and its multimers. Female, but not male, FCH patients had significant lower total
adiponectin and both HMW and low molecular weight (LMW) adiponectin than controls. The adiponectin sensitivity index (ASI), reflected by HMW/total adiponectin, and the LMW/HMW adiponectin ratio did not differ significantly between FCH females and control females. However, FCH females with CVD exhibited significantly lower ASI (34.2 +/- 10.1% vs 46.0 +/- 7.1%) and higher LMW/HMW ratio (1.5 +/- 0.8 vs 0.7 +/- 0.3) compared to FCH females without CVD, reflecting a more atherogenic adiponectin multimer distribution. (C) 2008 Elsevier Inc. All rights reserved.

3/7/6 (Item 6 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

0020549023 BIOSIS NO.: 200800595962

Familial aggregation in patients with non-alcoholic steatohepatitis
AUTHOR: Tokushige Katsutoshi (Reprint); Yatsuji Satoru; Hashimoto Etsuko;
Kabutake Ayae; Tobari Maki; Taniai Makiko; Shiratori Keiko
AUTHOR ADDRESS: Tokyo Womens Med Univ, Dept Med and Gastroenterol, Tokyo,
Japan**Japan
AUTHOR E-MAIL ADDRESS: ktoku@pg7.so-net.ne.jp
JOURNAL: Internal Medicine (Tokyo) 47 (5): p405-410 2008 2008
ITEM IDENTIFIER: doi:10.2169/internalmedicine.47.0476
ISSN: 0918-2918
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: We encountered three families that showed NASH accumulation. In
family #1, a 21-year-old son and 10-year-old daughter were diagnosed with
nonalcoholic steatohepatitis (NASH). They shared two adiponectingene
single nucleotide polymorphisms (SNP). In family #2, a 51-year-old mother
and 27-year-old son were diagnosed with NASH and shared the SNPs of other
genes. In family #3, a 66-year-old mother and 34-year-old son were
diagnosed with NASH and shared the SNPs of other genes. SNP sites
differed among the three families, suggesting that the genes associated
with the occurrence of NASH might be different in each patient.

3/7/7 (Item 7 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

0020485825 BIOSIS NO.: 200800532764
Adipocyte adhesion molecule (ACAM) inhibits adipocyte hypertrophy in obesity
AUTHOR: Murakami Kazutoshi (Reprint); Wada Jun; Nakatsuka Atsuko; Kanzaki
Motoko; Teshigawara Sanae; Terami Takahiro; Inoue Kentaro; Makino Hirofumi
AUTHOR ADDRESS: Okayama, Japan**Japan
JOURNAL: Diabetes 57 (Suppl. 1): pA110-A111 JUN 2008 2008
CONFERENCE/MEETING: 68th Annual Meeting of the American-Diabetes-Association San Francisco, CA, USA June 06 -10, 2008;
20080606
SPONSOR: Amer Diabet Assoc
ISSN: 0012-1797
DOCUMENT TYPE: Meeting; Meeting Abstract
RECORD TYPE: Citation

LANGUAGE: English

3/7/8 (Item 8 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

0020476411 BIOSIS NO.: 200800523350
The effects of phytosteryl ferulates on multimeric form of adiponectin secreted from 3T3-L1 adipocytes
AUTHOR: Ohara K (Reprint); Nagasaka R; Ushio H
AUTHOR ADDRESS: Tokyo Univ Marine Sci and Technol, Tokyo, Japan**Japan
JOURNAL: FEBS Journal 275 (Suppl. 1): p140 JUN 2008 2008
CONFERENCE/MEETING: Joint Conference of the 33rd FEBS Congress/11th IUBMB
Conference Athens, GREECE June 28 -July 03, 2008; 20080628
SPONSOR: Federat Biochem Soc
Int Union Biochem & Mole Biol
ISSN: 1742-464X_(print) 1742-4658_(electronic)
DOCUMENT TYPE: Meeting; Meeting Poster
RECORD TYPE: Citation
LANGUAGE: English

3/7/9 (Item 9 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

0020393277 BIOSIS NO.: 200800440216
High molecular mass multimer complexes and vascular expression contribute to high adiponectin in the fetus
AUTHOR: Pinar H; Basu S; Hotmire K; Laffineuse L; Presley L; Carpenter M;
Catalano P M; Hauguel-de Mouzon S (Reprint)
AUTHOR ADDRESS: Case Western Reserve Univ, Metrohlth Med Ctr, Dept Reprod
Biol, 2500 MetroHlth Dr, Cleveland, OH 44109 USA**USA
AUTHOR E-MAIL ADDRESS: shdemouzon@metrohealth.org
JOURNAL: Journal of Clinical Endocrinology & Metabolism 93 (7): p2885-2890
JUL 1 2008 2008
ITEM IDENTIFIER: doi:10.1210/jc.2008-0009
ISSN: 0021-972X
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: Context: High plasma adiponectin concentrations in human fetuses and neonates are unique features of early developmental stages.
Yet, the origins of the high adiponectin concentrations in the perinatal period remain elusive.Objective: This study was undertaken to

identify the sources and functional properties of adiponectin in utero.Design and Methods: Tissue specimens were obtained at autopsy from 21- to 39-wk-old stillborn human fetuses. Adipose tissue and placenta were obtained at term elective cesarean section. Adiponectin complexes and expression were measured by immunodetection and real-time PCR.Results: Adiponectin mRNA transcripts were detected in fetal sc and omental adipose depots at lower concentrations than in maternal adipose tissue. Immunoreactive adiponectin was also observed in vascular endothelial cells of fetal organs, including skeletal muscle, kidney, and brain. The absence of adiponectin in all placental cell types and lack of correlation between maternal and umbilical adiponectin indicate that umbilical adiponectin reflects its exclusive production by fetal tissues. The most prominent forms of adiponectin in fetal plasma were high and low molecular mass (HMW and LMW) multimers of 340 and 160 kDa, respectively. The proportion of the HMW complexes was 5-fold ($P < 0.001$) higher in umbilical plasma than in adult. The high HMW and total adiponectin levels were associated with lower insulin concentration and lower homeostasis model of assessment of insulin resistance indices in umbilical plasma, reflecting higher insulin sensitivity of the fetus compared with adult.Conclusions: The abundance of HMW adiponectin and its vascular expression are characteristics of human fetal adiponectin. Combined with high insulin sensitivity, fetal adiponectin may be a critical determinant of in utero growth.

3/7/10 (Item 10 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

0020369310 BIOSIS NO.: 200800416249
Family history and familial aggregation in patients with nonalcoholic steatohepatitis
AUTHOR: Noto Haruka; Tokushige Katsutoshi; Hashimoto Etsuko; Kabutake Ayae; Tobari Maki; Yatsuji Satoru; Taniai Makiko; Shiratori Keiko
JOURNAL: Gastroenterology 134 (4, Suppl. 1): pA784 APR 2008 2008
CONFERENCE/MEETING: Digestive Disease Week Meeting/109th Annual Meeting of the American-Gastroenterological-Association San Diego, CA, USA May 17 -22, 2008; 20080517
SPONSOR: Amer Gastroenterol Assoc
ISSN: 0016-5085

DOCUMENT TYPE: Meeting; Meeting Abstract
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: Genetic background contributes to the onset / progress of nonalcoholic steatohepatitis (NASH), as with other lifestyle-related diseases. In order to investigate the role of genetic background in NASH, we investigated the frequencies of diabetes mellitus (DM), hypertension (HT), and hyperlipidemia (HL) in the families of NASH patients. In addition, we found 3 families with NASH aggregation and investigated their single nucleotide polymorphisms (SNP). (Method) We examined 106 NASH patients and 37 control subjects. We compared the frequency of intrafamilial DM / HT/ HL by questionnaire. In three families with NASH aggregation, SNPs of TNF, adiponectin, beta 3-adrenergic receptor, interleukin- 1, MTP, MnSOD genes were examined. (Results)1. The frequency of DM in parents of NASH patients (27%) is significantly higher than that of parents of controls (7%). A trend toward DM was noted in the mothers, but not the fathers. The frequency of DM in brothers of NASH patients was also higher than that of controls. Regarding HT and HL, there was no difference between two groups. 2. In 106 NASH patients, we found three families that showed NASH accumulation. In family #1, a 21-year-old son and 10-year-old daughter were diagnosed with NASH and shared two adiponectin gene SNPs. In family #2, a 51-year-old mother and 27-year-old son were diagnosed with NASH and shared SNPs for TNF, beta 3-adrenergic receptor and MTP. In family #3, a 66-year-old mother and 34-year-old son were diagnosed with NASH and shared SNPs for MTP and MnSOD. SNP sites reported to be associated with NASH or DM, differed among the three families. in all patients, liver function was correlated with body weight. (Conclusion) Genomic background linked to DM might be related to the pathogenesis of NASH. However, critical SNP sites differed among the three families with NASH aggregation, suggesting that the genes associated with its occurrence might be different in each patient. In addition, if a person's genetic background is associated with the onset or progress of NASH, amelioration of living habits remains important.
? ds

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Set     Items   Description
S1      49381   ADIPONECTIN
S2        464   S1 AND (MULTIMER OR AGGREGAT?)
S3        110   RD S2  (unique items)
? t s3/7/11-110
>>>Format 7 is not valid in file 143

 3/7/11     (Item 11 from file: 5)
DIALOG(R)File   5:Biosis Previews(R)
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0020367692   BIOSIS NO.: 200800414631
The association of circulating adiponectin multimers with Barrett's
  esophagus
AUTHOR: Rubenstein Joel H; Kao John Y; Madanick Ryan D; Zhang Min;
Wang
  Meizhi; Spacek Melissa; Donovan Jena; Bright Stephanie D; Shaheen
  Nicholas J
JOURNAL: Gastroenterology  134 (4, Suppl. 1): pA437 APR 2008 2008
CONFERENCE/MEETING: Digestive Disease Week Meeting/109th Annual
Meeting of
the American-Gastroenterological-Association  San Diego, CA, USA  May
17
-22, 2008; 20080517
SPONSOR: Amer Gastroenterol Assoc
ISSN: 0016-5085
DOCUMENT TYPE: Meeting; Meeting Abstract
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: Background: Adiponectin is a peptide secreted from
  adipocytes that has insulin-sensitizing effects, is pro-apoptotic,
and is
  anti-inflammatory. Low circulating levels of adiponectin are
  associated with obesity, and have been associated with a number of
  epithelial cancers. A small pilot study suggested that low levels
may be
  associated with Barrett's esophagus (BE). The high molecular weight
(HMW)
  multimers of adiponectin are believed to be responsible lot its
  insulin-sensitizing effect, but the role of the various multimers in
  carcinogenesis is largely unknown. We sought to examine the
relationship
  of adiponectin with BE, including the relationship of the HMW
  multimers. Methods: We performed a clinic-based, cross-sectional
study of
  risk factors for the presence of BE. Controls were subjects
undergoing
  elective upper endoscopy for classic gastroesophageal reflux
symptoms
  (GER), who did not harbor endoscopic or histological evidence of BE.
  Cases were subjects with endoscopic evidence of BE and histological
```

confirmation of intestinalized metaplasia. The levels of plasma adiponectin (total and HMW) was measured using a commercially available ELISA kit (ALPCO Diagnostics, Salem NH). Adiponectin levels (total, HMW, and non-HMW) were compared between cases and controls using logistic regression. Adjustments were made for age, gender, race, smoking, and the presence of a hiatal hernia. Results: Plasma samples were obtained from 116 cases of BE, and 234 GER controls. Among controls, total, HMW, and non-HMW adiponectin were inversely correlated with body mass index (r = -0.24, p < 0.001; r = -0.20, p = 0.003; r = -0.23, p < 0.001, respectively), and torso/buttocks ratio (r = -0.22, p = 0.001; r = -0.17, p = 0.02 r = -0.22, p = 0.002, respectively). Lower levels of total adiponectin were only marginally associated with BE [1st tertile vs. 3rd tertile: unadjusted odds ratio (uOR) 1.36, 95% confidence interval (CI) 0.79, 2.36; adjusted odds ratio (aOR) 1.21,95% CI 0.60,2.45]. Higher levels of HMW adiponectin were also marginally associated with BE (3rd tertile vs. 1st tertile: uOR 1.57, 95% CI 0.88, 2.79, aOR 1.67, 95% CI 0.82, 3.38). Lower levels of non-HMW adiponectin were strongly associated with BE compared to GER controls (1st tertile vs. 3rd tertile: uOR 4.79, 95% CI 2.48, 9.23, aOR 4.30, 95% CI 1.93, 9.60; 2nd tertile vs. 3rd tertile: uOR 2.47, 95% CI 1.23, 4.95, aOR 2.13, 95% CI 0.96, 4.74). Conclusions: Low circulating levels of non-HMW adiponectin is strongly associated with the presence of BE compared to GER controls. Adiponectin may be active in the pathogenesis of BE, and may be useful in risk stratification for BE among those with GER symptoms.

3/7/12 (Item 12 from file: 5)

0020298117 BIOSIS NO.: 200800345056
Metabolic profile in sons of women with polycystic ovary syndrome
AUTHOR: Recabarren Sergio E; Smith Rosita; Rios Rafael; Maliqueo Manuel; Echiburu Barbara; Codner Ethel; Cassorla Fernando; Rojas Pedro; Sir-Petermann Teresa (Reprint)
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JOURNAL: Journal of Clinical Endocrinology & Metabolism 93 (5): p1820-1826
MAY 2008 2008
ITEM IDENTIFIER: doi:10.1210/jc.2007-2256
ISSN: 0021-972X
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: Context: Polycystic ovary syndrome (PCOS) is a common endocrine-metabolic disorder with strong familial aggregation. It has been demonstrated that parents and brothers of PCOS women exhibit insulin resistance and related metabolic defects. However, metabolic phenotypes in sons of PCOS women have not been described. Objective: Our objective was to assess the metabolic profiles in sons of women with PCOS during different stages of life: early infancy, childhood, and adulthood.Design: Eighty sons of women with PCOS (PCOSS) and 56 sons of control women without hyperandrogenism (C-S), matched for age, were studied. In early infancy, glucose and insulin were determined in the basal sample. In children and adults, a 2-h oral glucose tolerance test was performed with measurements of glucose and insulin. Adiponectin, leptin, C-reactive protein, SHBG, and serum lipids were determined in the basal sample during the three periods.Results: During early infancy, PCOSS showed higher weight (P = 0.038) and weight SD score (P = 0.031) than C-S. During childhood, weight (P = 0.003), body mass index (BMI) (P < 0.001), BMI SD score (P < 0.001), waist circumference (P = 0.001), total cholesterol (P = 0.007), and low-density lipoprotein cholesterol (P = 0.022) were higher in PCOSS compared with C-S, but after adjusting for BMI, these differences were nonsignificant. During adulthood, PCOSS exhibited higher weight (P = 0.022), BMI (P = 0.046), and waist circumference (P = 0.028) than C-S. Fasting insulin (P = 0.030), homeostasis model assessment for insulin resistance (P = 0.034), total cholesterol (P = 0.043), low-density lipoprotein cholesterol (P = 0.034), and 2-h insulin (P = 0.006) were also significantly higher and insulin sensitivity index composite significantly lower in PCOSS than in C-S (P =

0.003). After adjusting for BMI, only 2-h insulin and insulin sensitivity
index composite remained significantly different.Conclusions: This study
indicates that sons of PCOS women exhibit higher body weight from early
infancy. In addition, insulin resistance became evident as the subjects
got older, which may place them at risk for the development of type 2
diabetes and cardiovascular disease.

3/7/13 (Item 13 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

0020201323 BIOSIS NO.: 200800248262
Adiponectin multimer distribution, not absolute amount of plasma, correlates with depression severity in healthy elderly subjects
AUTHOR: Narita Kosuke; Murata Tetsuhito (Reprint); Hamada Toshihiko; Takahashi Tetsuya; Kosaka Hirotaka; Sudo Satoru; Mizukami Kimiko; Yoshida Haruyoshi; Wada Yuji
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JOURNAL: Progress in Neuro-Psychopharmacology & Biological Psychiatry 32 (
1): p124-127 JAN 1 2008 2008
ITEM IDENTIFIER: doi:10.1016/j.pnpbp.2007.07.022
ISSN: 0278-5846
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: Adiponectin is an adipocyte-specific secretory protein that circulates in serum as three oligomeric complexes known as the high, medium and low molecular weight form (HMW, MMW and LMW). HMW adiponectin has been suggested to be a better predictor of metabolic variables, and it was recently reported that the ratio of HMW to total adiponectin or to LMW, not the absolute amount of plasma adiponectin, might be crucial in determining insulin sensitivity. Insulin resistance (IR) is considered to be a primary component of vascular risk factors. Although the association of depression with atherosclerotic vascular diseases has been well documented, the contribution of IR to the evolution and progression of depression-associated vascular morbidity and mortality remains unknown.
The current preliminary study showed that the ratio of HMW to total

adiponectin or to LMW, not the absolute amount of plasma adiponectin, was negatively associated with depression severity in healthy elderly subjects without metabolic syndrome. This pilot study supports a promising role of adiponectin multimer distribution for clarifying the pathophysiological mechanism by which depression is associated with increased risk for IR, leading to cardiovascular disease, metabolic syndrome or type 2 diabetes. (c) 2007 Published by Elsevier Inc.

3/7/14 (Item 14 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

0020194083 BIOSIS NO.: 200800241022
Vascular effects of adiponectin: molecular mechanisms and potential therapeutic intervention
AUTHOR: Zhu Weidong; Cheng Kenneth K Y; Vanhoutte Paul M; Lam Karen S L; Xu Aimin (Reprint)
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JOURNAL: Clinical Science (London) 114 (5-6): p361-374 MAR 2008 2008
ITEM IDENTIFIER: doi:10.1042/CS20070347
ISSN: 0143-5221
DOCUMENT TYPE: Article; Literature Review
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: Adiponectin is a major adipocyte-secreted adipokine abundantly present in the circulation as three distinct oligomeric complexes. In addition to its role as an insulin sensitizer, mounting evidence suggests that adiponectin is an important player in maintaining vascular homoeostasis. Numerous epidemiological studies based on different ethnic groups have identified adiponectin deficiency (hypoadiponectinaemia) as an independent risk factor for endothelial dysfunction, hypertension, coronary heart disease, myocardial infarction and other cardiovascular complications. Conversely, elevation of circulating adiponectin concentrations by either genetic or pharmacological approaches can alleviate various vascular dysfunctions in animal models. Adiponectin exerts its vasculoprotective effects through its direct actions in the vascular system, such as increasing

endothelial NO production, inhibiting endothelial cell activation and endothelium-leucocyte interaction, enhancing phagocytosis, and suppressing macrophage activation, macrophage-to-foam cell transformation and platelet aggregation. In addition, adiponectin reduces neointima formation through an oligomerization-dependent inhibition of smooth muscle proliferation. The present review highlights recent research advances in unveiling the molecular mechanisms that underpin the vascular actions of adiponectin and discusses the potential strategies of using adiponectin or its signalling pathways as therapeutic targets to combat obesity-related metabolic and vascular diseases.

3/7/15 (Item 15 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

0020151494 BIOSIS NO.: 200800198433
High-molecular adiponectin as a marker of coronary artery disease
AUTHOR: Inoue Teruo (Reprint); Kotooka Norihiko; Komoda Hiroshi; Node Koichi
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JOURNAL: Circulation 116 (16, Suppl. S): p321 OCT 16 2007 2007
CONFERENCE/MEETING: 80th Annual Scientific Session of the American-Heart-Association Orlando, FL, USA November 04 -07, 2007; 20071104
SPONSOR: Amer Heart Assoc
ISSN: 0009-7322
DOCUMENT TYPE: Meeting; Meeting Abstract
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: Background: Adiponectin is an adipocyte-specific secretory protein that is highly and specifically expressed in adipose tissue. Plasma adiponectin. level is decreased in obese individuals, to be negatively correlated with visceral fat accumulation and the lower level of adiponectin has been suggested to be associated with coronary artery disease, especially with the development of acute coronary syndrome. In human plasma, adiponectin circulates as a trimer, a hexamer and a high-molecular weight multimer. High-molecular weight (HMW) adiponectin of 420kDa is suggested to be more important as to vascular protective activities than total amount of adiponectin. However, clinical significance of the plasma HMW adiponectin in cooronary artery disease is not evident. Methods and Results: We measured HMW adiponectin level in 149 patients undergoing diagnostic coronary angiography, suspected of chronic coronary artery disease. The

high molecular adiponectin level was lower in patients with vasospastic angina (3.4 +/- 2.4 mu g/dl, P<0.01), stable effort angina (3.3 +/- 2.6, P<0.001) and old myocardial infarction (3.8 +/- 2.9, P<0.01), compared to chest pain syndrome patients (control) (6.6 +/- 5.4). The level was lower in patients with multi-vessel disease (3.4 +/- 2.4 mu g/dl), compared to patients with single vessel disease (4.2 +/- 2.7, P<0.05) or no organic stenosis (5.1 +/- 4.5, P<0.01). During observation of 7 years' follow-up, onset of cardiovascular events was seen in 50 patients (34%). Among various risk factors, diabetes (P=0.02), insulin resistance assessed by homeostasis model assessment (P=0.06), no statin use (P=0.08), high sensitive C reactive protein level (P=0.0012) and HMW adiponectin level (P=0.0037) could predict cardiovascular events in univariate logistic regression analysis. However, multiple logistic regression analysis using these parameters showed that only HMW adiponectin was an independent predictor of cardiovascular event (OR; 2,23, 95%CI; 1.06-4.69, P=0.035). Conclusion: HMW adiponectin may be not only a marker for severity of coronary artery disease but also a predictor of future cardiovascular events in patients with coronary artery disease.

3/7/16 (Item 16 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

0020148187 BIOSIS NO.: 200800195126
A comparative study of the prevalence of the metabolic syndrome and its components in type 2 diabetic patients in two Caribbean islands using the new International Diabetes Federation definition
AUTHOR: Ezenwaka C E (Reprint); Nwagbara E; Seales D; Okali F; Hussain S; Raja B; Wheeler V; Sell H; Avci H; Eckel J
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JOURNAL: Archives of Physiology and Biochemistry 113 (4-5): p202-210 OCT-DEC 2007 2007
ITEM IDENTIFIER: doi:10.1080/13813450701475201
ISSN: 1381-3455

DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: Background and aim: Tobago and Trinidad are two Caribbean islands with distinct genetic background and lifestyles; while Tobago is serene and a tourist centre, Trinidad is characterized by a hustling and bustling lifestyle. The study was aimed at determining and comparing the prevalence of the metabolic syndrome (MetS) and its critical components in type 2 diabetic patients using the new International Diabetes Federation (IDF) definition. Methods: Four hundred and thirteen (166 Tobago, 247 Trinidad) type 2 diabetic patients visiting 10 lifestyle disease clinics were studied. Blood pressure, anthropometric parameters (height, weight, body mass index and waist circumference) and overnight fasting blood samples were taken. Plasma glucose and serum triglycerides, total cholesterol, LDL- and HDL-cholesterol, insulin, and adiponectin were determined. Insulin resistance (IR) was determined using-the HOMA method.Results: The patients in Tobago were significantly older than patients in Trinidad ($p < 0.001$) but the duration of diabetes (9.4 +/- 0.5 vs. 11.1 +/- 0.7 yr), medications, generalized (31.7 vs. 38.8%) and central (78.5 vs. 83.7%) obesity were similar ($p > 0.05$). In comparison with patients in Tobago, diabetic patients in Trinidad, irrespective of gender, had significantly higher prevalence of IDF critical components such as raised BP, raised triglycerides and reduced HDL-cholesterol (all, $p < 0.001$). Thus, while more patients in Trinidad were diagnosed with MetS based on three or four components, more patients in Tobago were diagnosed based on two components ($p < 0.001$).Conclusions: There were high prevalence rates of the components of the MetS in both the islands of Tobago and Trinidad. Quantitatively, the aggregation of the components is higher in patients in Trinidad, which constitute greater risk for adverse cardiovascular outcome. Controlling central obesity should be the target in preventing MetS in the two islands.

0020057647 BIOSIS NO.: 200800104586
High molecular weight multimer form of adiponectin as a useful marker to evaluate insulin resistance and metabolic syndrome in Japanese men
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JOURNAL: Metabolism Clinical and Experimental 56 (11): p1493-1499 NOV 2007
2007
ITEM IDENTIFIER: doi:10.1016/j.metabol.2007.06.015
ISSN: 0026-0495
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: Adiponectin is an adipocyte-specific secretory protein that possesses antidiabetic and anti atherosclerotic properties. Recent studies have demonstrated that the high molecular weight (HMW) multirner form is the active forrn of this protein. In patients with type 2 diabetes mellitus, HMW-total adiponectin ratio was reported to be a more useful marker than total adiponectin in the prediction of insulin resistance and metabolic syndrome. In the present study of healthy Japanese male subjects without any medication, we investigated the hypothesis that measuring only HMW adiponectin may be as effective as HMW-total ratio to predict insulin resistance and/or metabolic syndrome. This was a working community-based cross-sectional study of 637 mate subjects aged 30 to 65 years. Total and HMW adiponectin concentrations in serum were measured by enzyme-linked immunosorbent assay using commercially available kits. Serum HMW adiponectin level was inversely correlated with homeostasis model assessment of insulin resistance (HOMA-IR) (r = -0.375, P <.0001) even after adjustment for age and body mass index (r' = -0.245, P <.0001).

When we divided the study subjects into quartile groups with equal numbers of subjects, HOMA-IR in the 4 groups based on serum HMW adiponectin level was significantly different (P <.01). Metabolic syndrome score in the 4 groups based on serum HMW adiponectin level was also significantly different (P <.01). Area under the curve of receiver operator characteristic curves of HMW adiponectin (0.73) to evaluate the presence of insulin resistance (HOMA-IR >2.5) was larger

than that of total adiponectin (0.68) or HMW-total ratio (0.70). Area under the curve of receiver operator characteristic curves of HMW adiponectin (0.70) to evaluate the presence of metabolic syndrome (body mass index-based modified criteria) was also larger than that of total adiponectin (0.65), but equal to that of HMW-total ratio (0.70). These results suggest that simply measuring HMW adiponectin may be as effective as HMW-total ratio to evaluate the presence of insulin resistance and metabolic syndrome, at least in nondiabetic subjects who are not receiving any medication. (C) 2007 Elsevier Inc. All rights reserved.

3/7/18 (Item 18 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

0020026143 BIOSIS NO.: 200800073082
Multimers and adiponectin gene 276G > T polymorphism in the Japanese population residing in rural areas
AUTHOR: Ishibashi Kiyomi; Takamura Noboru (Reprint); Aoyagi Kiyoshi; Yamasaki Hironori; Abiru Norio; Nakazato Mio; Kamihira Shimeru; Maeda Takahiro
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JOURNAL: Clinical Chemistry and Laboratory Medicine 45 (11): p1457-1463
2007 2007
ITEM IDENTIFIER: doi:10.1515/CCLM.2007.313
ISSN: 1434-6621
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: Background: Although it has been shown that high-molecular weight adiponectin is an active form, few studies have attempted to clarify the relationship between high molecular weight adiponectin and markers linked with cardiovascular diseases in the general population.Methods: We screened 236 Japanese study participants recruited from the general population, residing in one large and four small islands. In addition to serum lipids and lipoproteins, serum total adiponectin and each multimer were measured. The genotype single-nucleotide polymorphism 276G > T was detected in real-time PCR with LightCycler (R) hybridization probes, using fluorescent-labeled

nucleotides.Results: Multiple linear regression analysis showed that high-molecular weight adiponectin, as well as total adiponectin, were significantly correlated with body weight, body mass index, high-density lipoprotein cholesterol and triglycerides. Total adiponectin and high-molecular weight adiponectin concentrations were not significantly different between GG and TX (GT and TT) genotypes of 276G > T polymorphism in the adiponectin gene. Interestingly, no differences were observed for participants from the large island between GG and TX genotypes with regard to both total adiponectin and high-molecular weight adiponectin, whereas significant differences were observed for those from the small islands.Conclusions: Our results show that total adiponectin and high-molecular weight adiponectin are associated with similar factors in the general population. Furthermore, different effects of 276G > T for participants from small and large islands suggest that regional background due to geographic barriers may control the effects of 276G > T on adiponectin concentrations.

3/7/19 (Item 19 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

0019999928 BIOSIS NO.: 200800046867
Total and high molecular weight but not trimeric or hexameric forms of adiponectin correlate with markers of the metabolic syndrome and liver injury in Thai subjects
AUTHOR: Liu Ying; Retnakaran Ravi; Hanley Anthony; Tungtrongchitr Rungsunn; Shaw Collin; Sweeney Gary (Reprint)
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JOURNAL: Journal of Clinical Endocrinology & Metabolism 92 (11): p 4313-4318 NOV 2007 2007
ITEM IDENTIFIER: doi:10.1210/jc.2007-0890
ISSN: 0021-972X
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: Context/Objective: Decreased total adiponectin has been associated with metabolic disorders, including obesity, diabetes, fatty liver, and the metabolic syndrome. Although circulating adiponectin is composed of trimers, hexamers, and high molecular weight (HMW)

multimers, there has been limited study of the specific metabolic correlates of these isoforms in humans. Thus, our objective was to evaluate the associations of these adiponectin isoforms with metabolic and anthropometric parameters.Design/Participants/Setting: A total of 53 diabetic and 68 nondiabetic subjects attending outpatient clinics underwent cross-sectional metabolic characterization. Circulating levels of HMW, hexameric, and trimeric adiponectin were measured using a multimeric adiponectin ELISA based upon selective protease-mediated digestion.Results: On Spearman univariate analysis, both total and HMW adiponectin levels were inversely associated with body mass index, fasting glucose, homeostasis model of assessment of insulin resistance, triglycerides, and alanine aminotransferase (ALT) (all vertical bar r vertical bar >= 0.22; P < 0.05), with the HMW isoform also positively correlated with high-density lipoprotein cholesterol (r = 0.19; P = 0.036). In contrast, hexameric and trimeric adiponectin were significantly associated with only body mass index (r = -0.23; P = 0.0102) and mid-upper arm circumference (r = 0.21; P = 0.039), respectively. On separate forward stepwise multiple linear regression analyses, fasting glucose and ALT emerged as independent, negative covariates of both total and HMW adiponectin, whereas no independent covariates of hexameric and trimeric adiponectin were identified. Furthermore, after adjustment for age, gender, and diabetes, mean ALT was highest in subjects in the lowest tertile of HMW adiponectin, followed in turn by the middle and highest tertiles, respectively (trend P = 0.028).Conclusions: HMW adiponectin, but not hexameric or trimeric, tracks with the metabolic correlates of total adiponectin. Furthermore, an independent inverse association exists between ALT and HMW adiponectin.

3/7/20 (Item 20 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

0019875706 BIOSIS NO.: 200700535447
Improved ELISA for selective measurement of adiponectin multimers and identification of adiponectin in human cerebrospinal fluid
AUTHOR: Ebinuma Hiroyuki; Miida Takashi (Reprint); Yamauchi Toshimasa; Hada Yusuke; Hara Kazuo; Kubota Naoto; Kadowaki Takashi

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JOURNAL: Clinical Chemistry 53 (8): p1541-1544 AUG 2007 2007
ITEM IDENTIFIER: doi:10.1373/clinchem.2007.085654
ISSN: 0009-9147
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: Background: Human serum adiponectin exists in 3 multimer forms: high molecular weight (HMW), middle molecular weight, and low molecular weight (LMW), with some of the latter bound to albumin (Alb)-LMW. Some studies have suggested that adiponectin crosses the blood-brain barrier and plays a central role in energy homeostasis.Methods: To determine cerebrospinal fluid (CSF) adiponectin at extremely low concentrations, we modified the protocol of the ELISA system used to assay serum adiponectin. The 3 multimers of adiponectin were measured separately by pretreating CSF with 2 proteases. We measured the CSF adiponectin concentrations in anonymous human samples (n = 19). The molecular sizes of adiponectin in CSF pretreated with proteases or untreated were determined by use of native PAGE and immunoblotting.Results: The ELISA system measured adiponectin in the range of 1.0-167 mu g/L. The between-assay imprecision estimates (CVs) were 6%-17% for the 3 forms. The mean total CSF adiponectin concentration (7.2 mu g/L) was similar to 1/ 1000 of the mean concentration in serum. Unlike serum adiponectin, the LMW and Alb-LMW forms predominated in all of the CSF samples. Immunoblotting analysis revealed that most LMW forms were bound to Alb, although the HMW form was detected in some samples.Conclusions: The modified ELISA system measures the 3 multimers separately and is sufficiently sensitive to measure adiponectin in CSF. (c) 2007 American Association for Clinical Chemistry.

3/7/21 (Item 21 from file: 5)

0019871880 BIOSIS NO.: 200700531621
Enhanced adiponectin multimer ratio and skeletal muscle adiponectin receptor expression following exercise training and diet in older insulin-resistant adults
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JOURNAL: American Journal of Physiology - Endocrinology and Metabolism 293
(1): pE421-E427 JUL 2007 2007
ITEM IDENTIFIER: doi:10.1152/ajpendo.00123.2007
ISSN: 0193-1849
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: Circulating adiponectin is reduced in disorders associated with insulin resistance. This study was conducted to determine whether an exercise/diet intervention would alter adiponectin multimer distribution and adiponectin receptor expression in skeletal muscle. Impaired glucose-tolerant older (> 60 yr) obese (BMI 30-40 kg/m(2)) men (n = 7) and women (n = 14) were randomly assigned to 12 wk of supervised aerobic exercise combined with either a hypocaloric (ExHypo, similar to 500 kcal reduction, n = 11) or eucaloric diet (ExEu, n = 10). Insulin sensitivity was determined by the euglycemic, (5.0 mM) hyperinsulmemic (40 mU(.)m(-2) (.)min(-1)) clamp. Adiponectin multimers [high (HMW), middle (MMW), and low molecular weight (LMW)] were measured by nondenaturing Western blot analysis. Relative quantification of adiponectin receptor expression through RT-PCR was determined from skeletal muscle biopsy samples. Greater weight loss occurred in ExHypo compared with ExEu subjects (8.0 +/- 0.6 vs. 3.2 +/- 0.6%, P < 0.0001). Insulin sensitivity improved postintervention in both groups (ExHypo: 2.5 +/- 0.3 vs. 4.4 +/- 0.5 mg(.)kg FFM(-1.)min(-1), and ExEu: 2.9 +/- 0.4 vs. 4.1 +/- 0.4 mg(.)kg FFM(-1.)min(-1), P < 0.0001). Comparison of multimer isoforms revealed a decreased percentage in MMW relative to HMW and LMW (P < 0.03). The adiponectin SA ratio (HMW/total) was increased following both interventions (P < 0.05) and correlated with the percent change in insulin sensitivity (P < 0.03). Postintervention adiponectin receptor mRNA expression was also significantly increased (AdipoR1 P < 0.03, AdipoR2 P < 0.02). These data suggest that part of the improvement in insulin sensitivity following

exercise and diet may be due to changes in the adiponectin oligomeric, distribution and enhanced membrane receptor expression.

3/7/22 (Item 22 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

0019817901 BIOSIS NO.: 200700477642
Ethnic differences in adiponectin multimer distribution and relationships with metabolic syndrome traits
AUTHOR: Lara-Castro Cristina; Doud Erin C; Munoz Julian; Hunter Gary R;
Gower Barbara A; Garvey W Timothy
JOURNAL: Diabetes 56 (Suppl. 1): pA361 JUN 2007 2007
CONFERENCE/MEETING: 67th Annual Meeting of the American-Diabetes-Association Chicago, IL, USA June 22 -26, 2007; 20070622
SPONSOR: Amer Diabet Assoc
ISSN: 0012-1797
DOCUMENT TYPE: Meeting; Meeting Poster
RECORD TYPE: Citation
LANGUAGE: English

3/7/23 (Item 23 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

0019760460 BIOSIS NO.: 200700420201
Adiponectin and the metabolic syndrome: mechanisms med ating risk for metabolic and cardiovascular disease
AUTHOR: Lara-Castro Cristina (Reprint); Fu Yuchang; Chung B Hong; Garvey W
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JOURNAL: Current Opinion in Lipidology 18 (3): p263-270 JUN 2007 2007
ISSN: 0957-9672
DOCUMENT TYPE: Article; Literature Review
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: Purpose of review Adiponectin is secreted exclusively by adipocytes, aggregates in multimeric forms, and circulates at high concentrations in blood. This review summarizes recent studies highlighting cellular effects of adiponectin and its role in human lipid metabolism and atherosclerosis.Recent findings Adiponectin is an important autocrine/paracrine factor in adipose tissue that modulates

differentiation of preadipocytes and favors formation of mature adipocytes. It also functions as an endocrine factor, influencing whole-body metabolism via effects on target organs. Adiponectin multimers exert differential biologic effects, with the high-molecular-weight multimer associated with favorable metabolic effects (i.e. greater insulin sensitivity, reduced visceral adipose mass, reduced plasma triglycerides, and increased HDL-cholesterol). Adiponectin influences plasma lipoprotein levels by altering the levels and activity of key enzymes (lipoprotein lipase and hepatic lipase) responsible for the catabolism of triglyceride-rich lipoproteins and HDL. It thus influences atherosclerosis by affecting the balance of atherogenic and antiatherogenic lipoproteins in plasma, and by modulating cellular processes involved in foam cell formation.Summary Recent studies emphasize the role played by adiponectin in the homeostasis of adipose tissue and in the pathogenesis of the metabolic syndrome, type 2 diabetes, and atherosclerosis. These pleiotropic effects make it an attractive therapeutic target for obesity-related conditions.

3/7/24 (Item 24 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

0019679949 BIOSIS NO.: 200700339690
Selective purification and characterization of adiponectin multimer species from human plasma
AUTHOR: Hada Yusuke; Yamauchi Toshimasa; Waki Hironori; Tsuchida Atsushi; Hara Kazuo; Yago Hirokazu; Miyazaki Osamu; Ebinuma Hiroyuki; Kadowaki Takashi (Reprint)
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ITEM IDENTIFIER: doi:10.1016/j.bbrc.2007.03.004
ISSN: 0006-291X
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LANGUAGE: English

ABSTRACT: Adiponectin is an adipocyte-derived hormone and known to form several species of multimer, however, the precise components

of each multimer have not been fully determined. We purified each multimer adiponectin selectively from human plasma and characterized them by affinity columns using anti-adiponectin, gelatin, or anti-albumin antibody and gel filtration. We found that adiponectin exists as four species of multimers in human plasma. According to their migrating mobility and N-terminal amino acid analysis, we defined them as a trimer, albumin-binding trimer, hexamer, and HMW. Low pH shifted HMW to hexamer, raising the possibility that HNIW is a 12 mer or larger multimer. We also showed that HMW had the highest binding activity to the membrane fractions of C2C12 myocytes and activated AMPK most potently. Our results indicate that adiponectin forms diverse multimer species and at least some of the functional properties are dependent on a multimer status.

3/7/25 (Item 25 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

0019598281 BIOSIS NO.: 200700258022
Adiponectin produced by lymphocytes inhibits granulopoiesis.
AUTHOR: Crawford Lisa J (Reprint); Peake Roy W; Price Susan; Morris Treen C ; Irvine Alexandra E
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JOURNAL: Blood 108 (11, Part 1): p374A NOV 16 2006 2006
CONFERENCE/MEETING: 48th Annual Meeting of the American-Society-of-Hematology Orlando, FL, USA December 09 -12, 2006;
20061209
SPONSOR: Amer Soc Hematol
ISSN: 0006-4971
DOCUMENT TYPE: Meeting; Meeting Poster
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: Previous studies by our group have shown that normal unstimulated lymphocytes produce a protein which inhibits colony formation ofgranulopoietic progenitors, but has no effect on erythroid progenitors. Therefore, this inhibitor was initially designated GIA (granulopoietic inhibitory activity). GIA was identified as a glycoprotein of approximately 30 kDa, with a pl of 7.9 - 8.4. Futhermore, we demonstrated that this inhibitor may have physiological significance in that its production is altered in patients with neutropenia. GIA has proved

difficult to characterise to date since it is produced in relatively low amounts although it has a high specific biological activity. Adiponectin is an adipokine reported to share many of the inhibitory characteristics of GIA and has been demonstrated to act as a negative regulator of hematopoiesis and immune response. This study aimed to determine whether GIA is adiponectin or if it represents an adiponectin-like molecule. Lymphocyte conditioned medium (LCM) from lymhocytes cultured at I X 10(6) cells/ml in HL- I minimal medium was used as a source of GIA. Inclusion of LCM as 10% of the top layer of agar in a myeloid colony assay inhibited growth of CFU-GM by 52 11 % (n=3), confirming the presence of the inhibitory activity. RNA and protein from lymphocytes and LCM harvested over a 7 day culture period were subsequently investigated for adiponectin expression. Western blot analysis demonstrated a distinct banding pattern in days 3-7 LCM corresponding to monomers, dimers, trimers and greater. This is consistent with adiponectin which circulates as a multimer of trimers. Characterisation of GIA at the transcript level confirmed that GIA is in fact adiponectin. The N-terminal collagenous domain, C terminal globular domain and full length adiponectin were amplified by RT-PCR analysis. Adiponectin is thought to be secreted exclusively from adipocytes and much of our current knowledge of this molecule relates to its metabolic functions. Our study provides evidence that adiponectin is also produced by lymphocytes and may play a role in the pathogenesis of neutropenia.

3/7/26 (Item 26 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

0019587970 BIOSIS NO.: 200700247711
Adiponectin, ghrelin, and leptin differentially influence human platelet and human vascular endothelial cell functions: Implication in obesity-associated cardiovascular diseases
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JOURNAL: European Journal of Pharmacology 558 (1-3): p7-13 MAR 6 2007 2007
ISSN: 0014-2999
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: A very strong epidemiological link exists between obesity, the metabolic syndrome, diabetes and diabetes-associated cardiovascular pathologies. For this reason the peripheral effects of the centrally-acting satiety adipokines, adiponectin and leptin, and of non-adipose-derived hormones with similar effects, like ghrelin, have received considerable attention. In this report, we have extended our previous studies of the prothrombotic effects of leptin and determined the effects of adiponectin or ghrelin on human platelet activation. Thus, while leptin stimulated human platelet aggregation and adhesion, addition of adiponectin or of ghrelin did not affect either aggregation or adhesion of these cells; even at supraphysiological concentrations. In addition, we compared the impact of these three important hormones on microvascular endothelial cell permeability, an important parameter of endothelial function that when impaired contributes to several vascular pathologies. While physiologically relevant concentrations of either leptin or adiponectin increased the integrity of the diffusion barrier formed by a monolayer of human microvascular endothelial cells, only supra-physiological concentrations of ghrelin had this effect. None of these agents reduced microvascular endothelial barrier function. Taken together, our data are consistent with the ideas that leptin activates human platelets and limits transendothelial cell diffusion but that adiponectin only influences endothelial cell permeability. In contrast, ghrelin had neither of these effects. We propose that these data identify important differences in the effects of leptin, adiponectin or ghrelin on microvascular endothelial cells and platelets and may provide a basis on which to pharmacologically manipulate the selective effects of these peptides on these cell types in human cardiovascular or thrombotic diseases associated with obesity. (c)

3/7/27 (Item 27 from file: 5)

0019560653 BIOSIS NO.: 200700220394
Increased basal platelet activity, plasma adiponectin levels, and diabetes mellitus are associated with poor platelet responsiveness to in vitro effect of aspirin
AUTHOR: Takahashi Shinichi; Ushida Miho; Komine Risa; Shimizu Aya; Uchida Toshihiro; Ishihara Hiroaki (Reprint); Shibano Toshiro; Watanabe Gentaro; Ikeda Yasuo; Murata Mitsuru
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JOURNAL: Thrombosis Research 119 (4): p517-524 2007 2007
ISSN: 0049-3848
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: Introduction: Aspirin is one of the most effective antiplatelet agents and is now commonly used to prevent vascular events. In some patients, however, recurrent vascular events have been demonstrated despite aspirin therapy. Our objective was to characterize individuals showing poor response to in vitro effect of aspirin, using PFA-100.Methods: One hundred sixty-eight healthy male subjects were analyzed. We assessed platelet function tests, including PFA-100, whole blood aggregation, and optical platelet aggregation. Also measured were hemostatic and other parameters including von Wiltebrand factor (VWF:Ag), VWF ristocetin cofactor activity (VWF:RCo), soluble vascular adhesion molecule-1 (sVCAM-1), high sensitive C-reactive protein (hs-CRP), and adiponectin. Poor responders were defined as having a cottagen/epinephrine-induced closure time (CEPI-CT) under 250 s with PFA-100 when incubated with 10 mu M aspirin, whereas good responders were defined as having a CEPI-CT of more than 250 s.Results and conclusions: PFA-100 tests revealed that 40 subjects (24%) were poor responders (PR) and 128 (76%) were good responders (GR). Poor responsiveness was significantly associated with (1) higher basal platelet activities in PFA-100, as well as in whole blood aggregation and aggregometer;(2) increased level of adiponectin (8.8 +/- 4.1 mu g/mL [PR] vs 7.3 +/-

2.9 mu g/mL [GR], p=0.010);and (3) the presence of diabetes mellitus (17.5% [PR] vs 4.7% [GR], p=0.009). Importantly, whereas 24% of the subjects showed insufficient inhibition in PFA-100 when incubated with 10 PM aspirin, almost at[subjects showed maximum inhibition with 30 mu M aspirin. These observations suggest that higher doses of aspirin might overcome aspirin resistance. (c) 2006 Elsevier Ltd. All rights reserved.

3/7/28 (Item 28 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

0019452627 BIOSIS NO.: 200700112368
A patient with Werner syndrome and adiponectin gene mutation
AUTHOR: Hashimoto Naotake (Reprint); Hatanaka Sachiko; Yokote Koutaro; Kurosawa Hiroko; Yoshida Tomohiko; Iwai Rie; Takahashi Hidenori; Yoshida Katsuya; Horie Atsuya; Sakurai Kenichi; Yagui Kazuo; Saito Yasushi; Yoshida Shouji
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JOURNAL: Diabetes Research and Clinical Practice 75 (1): p27-29 JAN 2007
2007
ISSN: 0168-8227
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: Werner syndrome is a premature aging disease characterized by genomic instability and increased cancer risk. Here, we report a 45-year-old diabetic man as the first Werner syndrome patient found to have an adiponectin gene mutation. Showing graying and loss of hair, skin atrophy, and juvenile cataract, he was diagnosed with Werner syndrome type 4 by molecular analysis. His serum adiponectin concentration was low. In the globular domain of the adiponectin gene, I164T in exon 3 was detected. When we examined effects of pioglitazone (15 mg/day) on serum adiponectin multimer and monomer concentrations using selective assays, the patient's relative percentage increased in adiponectin concentration was almost same as that in the 18 diabetic patients without an adiponectin mutation, but the absolute adiponectin concentration was half of those seen in diabetic patients treated with the same pioglitazone dose

who had no adiponectin mutation. The response suggested that pioglitazone treatment might help to prevent future Werner syndrome-related acceleration of atherosclerosis. Present and further clinical relevant to atherosclerosis in this patient should be imformative concerning the pathogenesis and treatment of atherosclerosis in the presence of hypoadiponectinemia and insulin resistance. (c) 2006 Elsevier Ireland Ltd. All rights reserved.

3/7/29 (Item 29 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

0019448154 BIOSIS NO.: 200700107895
Serum concentrations of adiponectin and characterization of adiponectin protein complexes in dogs
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JOURNAL: American Journal of Veterinary Research 68 (1): p57-62 JAN 2007
2007
ISSN: 0002-9645
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: Objective-To assess serum concentrations of adiponectin and characterize adiponectin protein complexes in healthy dogs.Animals-11 healthy dogs.Procedures-Sera collected from 10 dogs were evaluated via velocity sedimentation and ultracentrifugation, SDS-PAGE, western immunoblotting, and radioimmunoassay, Visceral adipose tissue (approx 90 g) was collected from the falciform ligament of a healthy dog undergoing elective ovariohysterectomy, and adiponectin gene expression was assessed via a real-time PCR procedure.Results-Adiponectin gene expression was detected in visceral adipose tissue. Serum adiponectin concentrations ranged from 0.85 to 1.5 mu g/ml (mean concentration, 1.22 mu g/mL). In canine serum, adiponectin was present as a multimer, consisting of a low-molecular-weight complex (180 kd); as 3 (180-, 90-, and 60-kd) complexes under denaturing conditions; as 2 (90- and 60-kd) complexes

under reducing conditions; and as a dimer, a monomer, and globular head
region (60, 30, and 28 kd, respectively) under reducing-denaturing conditions. It is likely that adiponectin also circulates as a high-molecular-weight (360- to 540-kd) complex in canine serum, but resolution of this complex was not possible via SDS-PAGE.Conclusions and
Clinical Relevance-After exposure to identical experimental conditions,
adiponectin protein complexes in canine serum were similar to those detected in human and rodent sera. Circulating adiponectin concentrations in canine serum were slightly lower than concentrations in
human serum. Adiponectin gene expression was identified in canine visceral adipose tissue. Results suggest that adiponectin could be used as an early clinical marker for metabolic derangements, including
obesity, insulin resistance, and diabetes mellitus in dogs.

3/7/30 (Item 30 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

19410850 BIOSIS NO.: 200700070591
Circulating concentrations of high-molecular-weight adiponectin are increased following Roux-en-Y gastric bypass surgery
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JOURNAL: Diabetologia 49 (11): p2552-2558 NOV 2006 2006
ISSN: 0012-186X
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: Aims/hypothesis In addition to weight loss, bariatric surgery for
severe obesity dramatically alleviates insulin resistance. In this study,
we investigated whether circulating concentrations of the high-molecular-weight (HMW) form of adiponectin are increased following gastric bypass surgery. The HMW form is implicated as the multimer responsible for adiponectin's hepatic
insulin-sensitising actions.Subjects and methods We studied 19 women who
were undergoing Roux-en-Y gastric bypass surgery. Studies were conducted

prior to, and 1 and 12 months after surgery.Results One month after surgery, total plasma adiponectin concentrations were unchanged. Nevertheless, increases in both HMW (by 40 +/- 15%, p=0.006) and the proportion of adiponectin in the HMW form (from 40 +/- 2 to 50 +/- 2%, p < 0.0001) were observed. At 12 months, total and HMW adiponectin concentrations were increased by 58 +/- 8% and 118 +/- 21%, respectively (both p < 0.001). The majority (80%) of the increase of total adiponectin was due to an increase of the HMW form. After adjustment for covariates, increases of HMW and total adiponectin at 12 months were correlated with the decrease of fat mass (HMW, p=0.0076; total, p=0.0302). In subjects with improved insulin sensitivity at 12 months after surgery (n=18), the increase of HMW, but not that of total adiponectin, predicted the relative decrease of insulin resistance (HMW: p=0.0044; total: p=0.0775, after adjustment for covariates).Conclusions/interpretation These data suggest that the reduction of fat mass following gastric bypass surgery is an important determinant of the increase of HMW adiponectin concentrations, which in turn is associated with and may contribute to the resulting improvement of insulin sensitivity.

3/7/31 (Item 31 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

19407881 BIOSIS NO.: 200700067622
Increased hypothalamic 5-HT2A receptor gene expression and effects of pharmacologic 5-HT2A receptor inactivation in obese A(y) mice
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JOURNAL: Biochemical and Biophysical Research Communications 351 (4): p
1078-1082 DEC 29 2006 2006
ISSN: 0006-291X
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: Serotonin (5-hydroxytryptamine; 5-HT) 2A receptors contribute to the effects of 5-HT on platelet aggregation and vascular smooth muscle cell proliferation, and are reportedly involved in decreases in

plasma levels of adiponectin, an adipokine, in diabetic subjects. Here, we report that systemic administration of sarpogrelate, a 5-HT2A receptor antagonist, suppressed appetite and increased hypothalamic pro-opiomelanocortin and cocaine- and amphetamine-regulated transcript, corticotropin releasing hormone, 5-HT2C, and 5-HT1B receptor gene expression. A(y) mice, which have ectopic expression of the agouti protein, significantly increased hypothalamic 5-HT2A receptor gene expression in association with obesity compared with wild-type mice matched for age. Systemic administration of sarpogrelate suppressed overfeeding, body weight gain, and hyperglycemia in obese A(y) mice, whereas it did not increase plasma adiponectin levels. These results suggest that obesity increases hypothalamic 5-HT2A receptor gene expression, and pharmacologic inactivation of 5-HT2A receptors inhibits overfeeding and obesity in A(y) mice, but did not increase plasma adiponectin levels.

3/7/32 (Item 32 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

19321165 BIOSIS NO.: 200600666560
Platelet activation is associated with hypoadiponectinemia and carotid atherosclerosis
AUTHOR: Shoji Takuhito; Koyama Hidenori (Reprint); Fukumoto Shinya; Maeno Takaaki; Yokoyama Hisayo; Shinohara Kayo; Emoto Masanori; Shoji Tetsuo; Yamane Takahisa; Hino Masayuki; Shioi Atsushi; Nishizawa Yoshiki
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JOURNAL: Atherosclerosis 188 (1): p190-195 SEP 2006 2006
ISSN: 0021-9150
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RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: Adiponectin, an adipokine secreted specifically from adipose tissue, has plurifunctions including antidiabetic, antiatherosclerotic, and antiinflammatory functions. Recently, platelet activation and the subsequent local inflammation have been implicated in progression of atherosclerosis. The aim of the study is to examine the interrelation among plasma adiponectin levels, platelet activation

status and quantitatively determined carotid atherosclerosis. Subjects (n = 277)including 136 type 2 diabetic, 138 hypertensive, and 203 hypercholesterolemic patients participated in the study. Platelet activation was determined as percentage of polymorphonuclear cells (PMNs) or monocytes aggregated with platelets analyzed by CD41-positivity determined by whole-blood flow cytometry. PMN-platelet aggregates were significantly and positively associated with carotid atherosclerosis (intimal-medial thickness, IMT) with the interaction stronger than that of monocyte-platelet aggregates. Stepwise regression analyses revealed that PMN-platelet aggregates were the third strongest determinant of carotid IMT, with age and HbA I c stronger independent determinants. Simple and stepwise regression analyses of the factors associated with PMN-platelet'aggregates revealed that HbA I c (r = 0.423), serum adiponectin levels (r = -0.289) and age (r = -0.184) were the three independent determinants. Thus, our data unveil novel link between hypoadiponectinemia and platelet activation. (c) 2005 Elsevier Ireland Ltd. All rights reserved.

19228177 BIOSIS NO.: 200600573572
A novel ELISA system for selective measurement of human adiponectin multimers by using proteases
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JOURNAL: Clinica Chimica Acta 372 (1-2): p47-53 OCT 2006 2006
ISSN: 0009-8981
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RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: Background: Adiponectin, an antiatherogenic adipocyte-derived protein exists in human blood as multiple isoforms-trimeric low molecular weight (LMW), albumin-binding LMW (Alb-LMW), hexameric middle molecular weight (MMW), and high molecular weight (HMW) forms. We developed a novel ELISA system to detect total

human adiponectin and the selective level of each adiponectin multimer for investigating the distribution of these levels in human blood.Methods: Two monoclonal antibodies that were raised against human adiponectin were used to construct a sandwich ELISA to measure adiponectin levels. Adiponectin multimers were selectively measured after sample pretreatment with two proteases that specifically digested the trimeric forms or both the hexameric and trimeric forms.Results: The ELISA had a dynamic range of 0.075-4.8 ng/ml. Intraassay variations (CV) were 5.3% (total adiponectin), 4.1% (MMW+HMW), and 3.3% (HMW). Comparison of the results of ELISA and quantitative western blot analysis of multimeric adiponectin in serum samples revealed good correlation (LMW+Alb-LMW, r = 0.873; MMW, r = 0.907; HMW, r = 0.950). Each of the three forms of adiponectin multimer levels closely correlated with total adiponectin levels in healthy subjects.Conclusions: This ELISA system can be used to further investigate the physiological roles of human adiponectin multimers. (c) 2006 Elsevier B.V. All rights reserved.

3/7/34 (Item 34 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

19192087 BIOSIS NO.: 200600537482
Adiponectin added into the plasma of healthy probands does not affect platelet aggregability
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JOURNAL: Biomedical Papers (Olomouc) 150 (1): p89-90 JUL 2006 2006
ISSN: 1213-8118
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: Six healthy non-obese probands without medical therapy and history of disease were tested. In all of them platelet aggregability with addition of human recombinant adiponectin in different concentrations (100; 75; 50 and 25 ng/l) were measured. It is concluded that increased level of adiponectin has no significant antiaggregation effect on platelets from individuals without hypoadiponectinemia.

3/7/35 (Item 35 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

19130379 BIOSIS NO.: 200600475774
Adiponectin multimerization is dependent on conserved lysines in the collagenous domain: Evidence for regulation of multimerization by alterations in posttranslational modifications
AUTHOR: Richards Ayanthi A; Stephens Tim; Charlton Hayley K; Jones Alun;
MacDonald Graeme A; Prins Johannes B; Whitehead Jonathan P (Reprint)
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JOURNAL: Molecular Endocrinology 20 (7): p1673-1687 JUL 2006 2006
ISSN: 0888-8809
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: Adiponectin is a secreted, multimeric protein with insulin-sensitizing, antiatherogenic, and antiinflammatory properties.
Serum adiponectin consists of trimer, hexamer, and larger high-molecular-weight (HMW) multimers, and these HMW multimers appear to
be the more bioactive forms. Multimer composition of adiponectin appears to be regulated; however, the molecular mechanisms involved are unknown. We hypothesize that regulation of adiponectin multimerization and secretion occurs via changes in posttranslational modifications (PTMs). Although a structural role for
intertrimer disulfide bonds in the formation of hexamers and HMW multimers is established, the role of other PTMs is unknown. PTMs identified in murine and bovine adiponectin include hydroxylation of multiple conserved proline and lysine residues and glycosylation of
hydroxylysines. By mass spectrometry, we confirmed the presence of these
PTMs in human adiponectin and identified three additional hydroxylations on Pro71, Pro76, and Pro95. We also investigated the role
of the five modified lysines in multimer formation and secretion of recombinant human adiponectin expressed in mammalian cell lines. Mutation of modified lysines in the collagenous domain prevented formation of HMW multimers, whereas a pharmacological inhibitor of prolyl- and lysyl-hydroxylases, 2,2'-dipyridyl, inhibited formation of
hexamers and HMW multimers. Bacterially expressed human adiponectin displayed a complete lack of differentially modified isoforms and failed
to form bona fide trimers and larger multimers. Finally, glucose-induced

increases in HMW multimer production from human adipose explants correlated with changes in the two-dimensional electrophoresis profile of adiponectin isoforms. Collectively, these data suggest that adiponectin multimer composition is affected by changes in PTM in response to physiological factors.

3/7/36 (Item 36 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

19126323 BIOSIS NO.: 200600471718
Olanzapine treatment is associated with reduced high molecular weight adiponectin in serum - A potential mechanism for olanzapine-induced insulin resistance in patients with schizophrenia
AUTHOR: Richards Ayanthi A; Hickman Ingrid J; Wang Amy Y-H; Jones Amanda L; Newell Felicity; Mowry Bryan J; Whitehead Jonathan P; Prins Johannes B; Macdonald Graeme A (Reprint)
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JOURNAL: Journal of Clinical Psychopharmacology 26 (3): p232-237 JUN 2006
2006
ISSN: 0271-0749
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: Treatment of schizophrenia with olanzapine and other atypical antipsychotic agents is associated with insulin resistance and diabetes mellitus. The mechanism for this is not understood. Adiponectin is an insulin-sensitizing cytokine secreted by adipocytes. It is present in serum in multimers of varying size. Trimers and hexamers are referred to as low molecular weight (LMW) adiponectin. Larger multimers (12-, 18-, and 24-mers) have been designated high molecular weight (HMW) adiponectin and seem responsible for the insulin-sensitizing action of this adipokine. The aim of this study was to examine total adiponectin and LMW and HMW multimers in serum from patients with schizophrenia treated with either olanzapine (n = 9) or other typical antipsychotics (n = 9) and compare results with 16 healthy sex-, body mass index-, and age-matched controls. The effects of olanzapine on adiponectin protein expression and secretion in in

vitro-differentiated primary human adipocytes were also examined. Patients receiving olanzapine had significantly lower total serum adiponectin as compared with those on conventional treatment and controls (5.23 +/- 1.53 ng/mL vs. 8.20 +/- 3.77 ng/mL and 8.78 +/- 3.8 ng/mL; P < 0.05 and P < 0.01, respectively). The HMW adiponectin was also reduced in patients on olanzapine as compared with the disease and healthy control groups (1.67 +/- 0.96 ng/mL vs. 3.87 +/- 2.69 ng/mL and 4.07 +/- 3.2 ng/mL; P < 0.05 for both). The LMW adiponectin was not different between patient groups (P = 0.15) but lower in patients on olanzapine as compared with controls (3.56 +/- 10.85 ng/mL vs. 4.70 +/- 1.4 ng/mL; P < 0.05). In vitro, short duration (up to 7 days) olanzapine exposure had no effect on total adiponectin expression or multimer composition of secreted protein. In summary, this study demonstrates a correlation between olanzapine treatment and reduced serum adiponectin, particularly HMW multimers. This may not be a direct effect of olanzapine on adipocyte expression or secretion of adiponectin. These observations provide insights into possible mechanisms for the association between olanzapine treatment and insulin resistance.

3/7/37 (Item 37 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

19072283 BIOSIS NO.: 200600417678
Identification of amino-terminal region of adiponectin as a physiologically functional domain
AUTHOR: Ujiie Hidetoshi; Oritani Kenji (Reprint); Kato Hisashi; Yokota Takafumi; Takahashi Isao; Maeda Tetsuo; Masaie Hiroaki; Ichii Michiko; Kamada Yoshihiro; Tamura Shinji; Kihara Shinji; Funahashi Tohru; Tomiyama Yoshiaki; Kanakura Yuzuru
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JOURNAL: Journal of Cellular Biochemistry 98 (1): p194-207 MAY 1 2006 2006
ISSN: 0730-2312
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: Adiponectin is an abundant adipose-specific protein, which acts as an anti-diabetic, anti-atherogenic, and anti-inflammatory adipokine. Although recent advances in the field of adiponectin have been made by the identification of adiponectin receptors and by the understanding about relationship between its multimerization and functions, detailed molecular background remains unclear. Our established anti-human adiponectin antibodies, ANOC 9103 and ANOC 9104, blocked some adiponectin functions Such as the growth inhibition of 13-lymphocytes on stromal cells and the inhibition of acetylated LDL uptake in macrophages, Suggesting that they may recognize important functional regions of adiponectin. As a result of epitope mapping based on the ability to bind to the deleted adiponectin Mutants, we identified that these antibodies recognize amino-terminal region of adiponectin before the beginning of the collagen-like domain. Notably, a peptide fragment (DQETTTQGPGVLLPLPKGACTGWMA) corresponding to amino acid residues 1741 of human adiponectin could bind to restricted types of cells and block adiponectin-induced cyclooxygenase-2 gene expression and prostaglandin E-2 production in MS-5 stromal cells. Moreover, the deletion of its amino-terminal region reduced the abilities to inhibit not only collagen-induced platelet aggregation but also diet-induced hepatic steatosis. These data indicate that amino-terminal region of adiponectin is a physiologically functional domain and that a novel receptor, which recognizes amino-terminal region of adiponectin, may exist on some types of cells. Further investigations will contribute to the understanding of molecular mechanisms about adiponectin functions as well as to the designing of novel strategies for the treatment of patients with insulin-resistance, vascular dysfunction, and chronic inflammation.

19061038 BIOSIS NO.: 200600406433
A novel enzyme-linked immunosorbent assay specific for high-molecular-weight adiponectin
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RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: Human plasma contains at least three forms of adiponectin: a trimer, a hexamer, and a high-molecular-weight (HMW) multimer. We purified HMW adiponectin from human plasma using its affinity to gelatin and obtained monoclonal antibodies against it. On Western blot analysis, the reactivity of these monoclonal antibodies was shown to be restricted to a non-heat-denatured form of adiponectin molecules. On heating, the collagen-like domain of adiponectin molecules became denatured, and thus the trimer form could not be maintained. From these, monoclonal antibodies against HMW adiponectin were suggested to react with the intact trimer of adiponectin. With these monoclonal antibodies, we developed a sandwich ELISA system for quantifying adiponectin in human serum. Its specificity was verified by analysis of serum fractions separated by gel-filtration chromatography, and our ELISA system was found to be HMW adiponectin-specific. With this novel ELISA, the HMW adiponectin concentrations were 8.4 +/- 5.5 mu g/ml (mean +/- SD) in healthy women and 6.2 +/- 3.6 mg/ml in healthy men. Also, serum with a lower HMW adiponectin concentration was shown to have a lower HMW ratio (i.e., HMW adiponectin/total adiponectin).

3/7/39 (Item 39 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

18816017 BIOSIS NO.: 200600161412
Adiponectin multimeric complexes and the metabolic syndrome trait cluster
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JOURNAL: Diabetes 55 (1): p249-259 JAN 2006 2006
ISSN: 0012-1797
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: Adiponectin circulates in human plasma mainly as a 180-kDa low molecular weight (LMW) hexamer and a high molecular weight (HMW) multimer of similar to 360 kDa. We comprehensively examined the

relationships between circulating levels of total adiponectin, adiponectin multimers, and the relative distribution (i.e., ratio) of multimeric forms with key features of the metabolic syndrome. Total adiponectin (r = 0.45), HMW (r = 0.47), LMW (r = 0.31), and HMW-to-total adiponectin ratio (r = 0.29) were significantly correlated with insulin-stimulated glucose disposal rate. Similarly, total (r = -0.30), HMW (r = -0.38), and HMW-to-total adiponectin ratio (r = -0.34) were correlated with central fat distribution but not with total fat mass or BMI. Regarding energy metabolism, although there were no effects on resting metabolic rate, total (r = 0.41) and HMW (r = 0.44) were associated with increasing rates of fat oxidation. HMW-to-total adiponectin ratio increased as a function of total adiponectin, and it was HMW quantity (not total or HMW-to-total adiponectin ratio or LMW) that was primarily responsible for all of these relationships. Impact on nuclear magnetic resonance lipoprotein subclasses was assessed. HMW and total adiponectin were correlated with decreases in large VLDL (r = -0.44 and -0.41); decreases in small LDL (r = -0.41 and -0.36) and increases in large LDL (r = 0.36 and 0.30) particle concentrations accompanied by increased LDL particle size (r = 0.47 and 0.39); and increases in large HDL (r = 0.45 and 0.37) and HDL particle size (r = 0.53 and 0.47). Most of these correlations persisted after adjustment for metabolic covariables. In conclusion, first, serum adiponectin is associated with increased insulin sensitivity, reduced abdominal fat, and high basal lipid oxidation; however, it is HAM quantity, not total or HMW-to-total adiponectin ratio, that is primarily responsible for these relationships. Second, reduced quantities of HMW independently recapitulate the lipoprotein subclass profile associated with insulin resistance after correcting for glucose disposal rate and BMI. Finally, HMW adiponectin is an important factor in explaining the metabolic syndrome.

3/7/40 (Item 40 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

18813474 BIOSIS NO.: 200600158869
Adiponectin acts as an endogenous antithrombotic factor

AUTHOR: Kato Hisashi; Kashiwagi Hirokazu; Shiraga Masamichi; Tadokoro Seiji
; Kamae Tsuyoshi; Ujiie Hidetoshi; Honda Shigenori; Miyata Shigeki; Ijiri
Yoshinobu; Yamamoto Junichiro; Maeda Norikazu; Funahashi Tohru; Kurata
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JOURNAL: Arteriosclerosis Thrombosis and Vascular Biology 26 (1): p224-230
JAN 2006 2006
ISSN: 1079-5642
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: Objective: Obesity is a common risk factor in insulin resistance
and cardiovascular diseases. Although hypoadiponectinemia is associated
with obesity-related metabolic and vascular diseases, the role of
adiponectin in thrombosis remains elusive.Methods and Results: We
investigated platelet thrombus formation in adiponectin knockout
(APN-KO) male mice (8 to 12 weeks old) fed on a normal diet. There was no
significant difference in platelet counts or coagulation parameters
between wild-type (WT) and APN-KO mice. However, APN-KO mice showed an
accelerated thrombus formation on carotid arterial injury with a He-Ne
laser (total thrombus volume: 13.36 +/- 4.25 x 10(7) arbitrary units for
APN-KO and 6.74 +/- 2.87 x 10(7) arbitrary units for WT; n = 10; P <
0.01). Adenovirus-mediated supplementation of adiponectin
attenuated the enhanced thrombus formation. In vitro thrombus formation
on a type I collagen at a shear rate of 250 s(-1), as well as platelet
aggregation induced by low concentrations of agonists, was enhanced
in APN-KO mice, and recombinant adiponectin inhibited the enhanced
platelet aggregation. In WT mice, adenovirus-mediated
overexpression of adiponectin additionally attenuated thrombus
formation.Conclusion: Adiponectin deficiency leads to enhanced
thrombus formation and platelet aggregation. The present study
reveals a new role of adiponectin as an endogenous antithrombotic
factor.

3/7/41 (Item 41 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

18672568 BIOSIS NO.: 200600017963
Adiponectin multimer ratio is increased following exercise and diet treatment in impaired glucose tolerance
AUTHOR: Nardi Ashley E (Reprint); Marchetti Christine M; Phillips Susan A;
Ciaraldi Theodore P; Kirwan John P
JOURNAL: Diabetes 54 (Suppl. 1): pA267 2005 2005
CONFERENCE/MEETING: 65th Annual Meeting of the American-Diabetes-Association San Diego, CA, USA June 10 -14, 2005; 20050610
SPONSOR: Amer Diabet Assoc
ISSN: 0012-1797
DOCUMENT TYPE: Meeting; Meeting Poster
RECORD TYPE: Citation
LANGUAGE: English

3/7/42 (Item 42 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

18574272 BIOSIS NO.: 200510268772
Enhanced platelet aggregation and thrombogenic tendency in adiponectin-deficient mice
AUTHOR: Kato Hisashi (Reprint); Kashiwagi Hirokazu; Shiraga Masamichi; Honda Shigenori; Miyat Shigeki; Yamamoto Junichiro; Kurata Yoshiyuki;
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JOURNAL: Blood 104 (11, Part 1): p228A-229A NOV 16 2004 2004
CONFERENCE/MEETING: 46th Annual Meeting of the American-Society-of-Hematology San Diego, CA, USA December 04 -07, 2004; 20041204
SPONSOR: Amer Soc Hematol
ISSN: 0006-4971
DOCUMENT TYPE: Meeting; Meeting Abstract
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: Adiponectin is a 30 kDa protein secreted specifically from adipocytes and structurally composed of two distinct domains, C-terminal
collagen-like domain andN-terminal complement C1q-like globular domain.

Adiponectin is abundantly present in plasma at high concentration ranging from 2 to 30 mu g/ml. The plasma levels of adiponectin decreased in patients with obesity and diabetes. Recently it has been demonstrated that adiponectin has an anti-atherogenic activity. Hypoadiponectinemia is an independent risk factor for coronary artery disease in men. However. the role of adiponectin in hemostasis and thrombosis still remains obscure. In this study, we examined its role in hemostasis and thrombosis using adiponectin-deficient (APN-KO) mice (Nat. Med. 2002 Maeda et al.). APN-KO mice were. fed by normal chaw and studied at 8-12 weeks old. There were nodifferences in platelet counts, PT, APTT and plasma fibrinogen levels betweenAPN-KO and Wild-Type mice. Neither Wild-Type nor APN-KO mice showed detectable atherosclerotic lesion in carotid artery as well as whole aorta. We examinedtail-bleeding times as a measure of primary hemostasis. The tail bleeding time was 96.9 +/- 34.9 seconds in APN-KO mice, which was shorter than that in wild type mice (130.9 +/- 52.1 seconds, n=30, p<0.05). We next Studied thrombus formation in mice carotid artery using He-Ne laser induced in vivo thrombus formation model. Thrombus formation was induced by the interaction of irradiated He-Ne laser with evans blue dye injected into blood flow. The thrombus volumesformed during 10 minutes were significantly larger in APN-KO mice (6.74 +/- 2.87 x 10(7) arbitrary units for wild-type v.s. 13.4 +/- 4.25 x 10(7) arbitrary units for APN-KO mice, n=10, p<0.01). Adenovirus-mediated supplement of adiponectin compensated for the thrombotic tendency in APN-KO mice. In order to clarify the effects of adiponectin on platelet functon, we performed ex vivo experiments. In platelet aggregation studies under stirring conditions using platelet-rich plasma, platelet aggregation induced by low concentrations of agonists (ADP 2.5 mu M, collagen 2.5 mu g/ml, PAR4 peptide 75 mu M)was enhanced in APN-KO mice. Again the adenovirus-mediated supplement of adiponectin compensated for the enhancement of platelet aggregation. We next studied the thrombus formation on collagen coated surface under flow conditions. The thrombus formation was enhanced in APN-KO mice under shear rate at 250s(-1). Our data provide a first evidence that adiponectin plays a role in hemostasis and thrombosis as a negative modulator of platelet function.

3/7/43 (Item 43 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

17990911 BIOSIS NO.: 200400361700
Effects of novel peptides derived from the acidic tail of synuclein (ATS)
on the aggregation and stability of fusion proteins
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JOURNAL: Protein Engineering Design & Selection 17 (3): p251-260 March
2004 2004
MEDIUM: print
ISSN: 1741-0126 _(ISSN print)
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: The acidic tail of alpha-synuclein (ATSalpha) has been shown to
protect the glutathione S-transferase (GST)-ATSalpha fusion protein from
environmental stresses, such as heat, pH and metal ions. In this study,
we further demonstrated that the introduction of ATSalpha into other
proteins, such as dehydrofolate reductase and adiponectin, renders
the fusion proteins resistant to heat-induced aggregation and that
the acidic tail of beta- or gamma-synuclein can also protect the fusion
proteins from heat-induced aggregation. Interestingly, the heat
resistance of GST-ATSalpha deletion mutants, which contain shorter
peptides derived from the highly charged regions of ATSalpha, was
approximately proportional to the number of added Glu/Asp residues.
However, the negative charges in the ATSalpha-derived peptides appear
insufficient to explain the extreme heat resistance of the fusion
proteins, since polyglutamates appeared to be much less effective than
the ATSalpha-derived peptides in conferring heat resistance on the fusion
proteins. These results suggest that not only the negatively charged
residues but also the specific amino acid sequence of ATSalpha play an
important role in conferring extreme heat resistance on the fusion
proteins. Furthermore, the heat-induced secondary structural changes and
thermal inactivation curves of GST-ATSalpha deletion mutants indicated

that the introduction of ATSalpha-derived peptides does not significantly
affect the intrinsic stability of the fusion proteins.

3/7/44 (Item 44 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

17944881 BIOSIS NO.: 200400315638
Mechanisms of early insulin-sensitizing effects of thiazolidinediones in
type 2 diabetes
AUTHOR: Tonelli Julia; Li Weijie; Kishore Preeti; Pajvani Utpal B; Kwon
Elize; Weaver Charles; Scherer Philipp E; Hawkins Meredith (Reprint)
AUTHOR ADDRESS: Div EndocrinolDept MedDiabet Res and Training Ctr, Albert
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JOURNAL: Diabetes 53 (6): p1621-1629 June 2004 2004
MEDIUM: print
ISSN: 0012-1797 _(ISSN print)
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: Whereas thiazolidinediones (TZDs) are known to rapidly improve
insulin action in animals, short durations of TZD therapy have never been
studied in humans. Among the many known actions of TZDs, increased
circulating levels of the high molecular weight (HMW) multimer of
adiponectin may be an important insulin-sensitizing mechanism. We
examined the effects of only 21 days of 45 mg of pioglitazone (P+) versus
placebo (P-) in nine subjects with type 2 diabetes (HbA1c, 10.9 +/- 0.6%;
BMI, 31.9 +/- 1.5 kg/m2). Total adiponectin levels increased by
approximately twofold in P+ in association with increased adipose tissue
gene expression. However, plasma free fatty acid and glucose levels were
unchanged, and there were only minimal changes in other "adipokines."
Glucose fluxes ((3-3H)glucose infusion) were measured during 6-h
euglycemic (5 mmol/l) "pancreatic clamp" studies
(somatostatin/glucagon/growth hormone) with stepped insulin levels.
Pioglitazone induced marked decreases in endogenous glucose production
(P+ = 0.9 +/- 0.1 vs. P- = 1.7 +/- 0.3 mg . kg-1 . min-1; P < 0.05) at

physiologic hyperinsulinemia (dollar sign50 muU/ml), which was highly
correlated with an increased ratio of HMW adiponectin/total levels
(r2 = 0.90). Maximal insulin stimulation (dollar sign400 muU/ml) revealed
pioglitazone-associated increases in glucose T take (P+ = 10.5 +/- 0.9
vs. P- = 8.9 +/- 0.8 mg . kg-1. min-1; P < 0.05), which did not correlate
with HMW or total adiponectin levels. Thus, only 21 days of
pioglitazone therapy improved insulin action in humans with type 2
diabetes. Increased abundance of the BMW adiponectin multimer
may contribute to the hepatic insulin-sensitizing effects of these
agents.

3/7/45 (Item 45 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

17877926 BIOSIS NO.: 200400246873
Adiponectin concentrations as a criterion of metabolic control in
persons with type 2 diabetes mellitus?
AUTHOR: Stejskal David (Reprint); Ruzicka Viktor (Reprint); Adamovska Sylva
(Reprint); Jurakova Renata (Reprint); Proskova Jitka (Reprint); Jedelsky
Ladislav (Reprint); Bartek Josef
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JOURNAL: Biomedical Papers (Olomouc) 147 (2): p167-172 December 2003 2003
MEDIUM: print
ISSN: 1213-8118 _(ISSN print)
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: Adiponectin (ADP) is an adipocytokin with many
antiatherogenic properties; its decreased level is associated with
numerous atherogenic diseases and syndromes (e.g. diabetes mellitus (DM),
dyslipidemia, endothelial dysfunction, hypertension, and obesity).
Decreased ADP values in blood may be an independent risk factor of
atherosclerotic (ATS) complications. Aim of the study: 1) Do persons with
type 2 diabetes have lower ADP values than individuals without DM but
with a high risk of ATS complications? 2) Do ADP values differ between
persons with well controlled and persons with uncontrolled type 2
diabetes? We examined 109 patients of the Metabolic Center of Hospital

Sternberk. Out of them, 58 had type 2 diabetes, others were individuals with variously expressed risk factors of early atherosclerosis (obesity, hypertension, age, family history, smoking, dyslipidemia, etc.). In all persons under this study the following parameters were determined in peripheral venous blood: adiponectin, resistin, leptin, ObRe, cholesterol, HDL-cholesterol, triacylglycerols, glucose, HbAlc, creatinine, urea, ALT, AST, CRP, homocysteine, thrombocyte aggregation after CPG induction. The whole group was divided according to the presence of type 2DM into two subgroups; persons with diabetes were divided into the well controlled and uncontrolled subgroups. All data obtained were processed statistically using the software SPSS for Windows and Medcalc. The adiponectin/BMI index correlated negatively with HbA1c value (correlation coefficient -0.37, p = 0.00053), triacylglycerols (-0.4, p = 0.000001), P-glucose (-0.3, p = 0.0017), uricemia (-0.35, p = 0.0007) and positively with HDL-cholesterol value (0.6, p=0.00001). Women had higher adiponectin values than men. Persons with hypertension and with diabetes mellitus, individuals with atherogenic lipotype or persons with inflammation signs had lower values than individuals without these diseases and syndromes. Persons with wellcontrolled diabetes mellitus had higher values than persons with uncontrolled diabetes (medians of the adiponectin/BMI index 9.7 vs. 6.7, p < 0.01). Persons with type 2 diabetes mellitus have lower ADP values than persons with a high ATS risk without diabetes mellitus. Persons with wellcontrolled diabetes mellitus (DM) and with satisfactory compensation have significantly higher ADP levels (independently of other metabolic parameters of DM control). ADP may be a new marker of metabolic control in persons with a high risk of atherosclerotic complications.

17700865 BIOSIS NO.: 200400081622
Role of disulfide bonds in Acrp30/adiponectin structure and signaling specificity. Different oligomers activate different signal transduction

pathways.
AUTHOR: Tsao Tau-Shuen; Tomas Eva; Murrey Heather E; Hug Christopher; Lee
David H; Ruderman Neil B; Heuser John E; Lodish Harvey F (Reprint)
AUTHOR ADDRESS: Whitehead Institute for Biomedical Research, 9 Cambridge
Center, Cambridge, MA, 02142, USA**USA
JOURNAL: Journal of Biological Chemistry 278 (50): p50810-50817 December
12, 2003 2003
MEDIUM: print
ISSN: 0021-9258
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: Acrp30/adiponectin is an adipocyte-derived serum protein with important roles in regulation of lipid and glucose metabolism, but which of its isoforms are biologically active remains controversial. We addressed this issue by first characterizing the structure of each individual Acrp30 oligomer and the determinants responsible for multimer formation. Freeze etch electron microscopy showed the trimer to exhibit a ball-and-stick-like structure containing a large globular sphere, an extended collagen stalk, and a smaller sphere on the opposite end of the stalk. The hexamer consists of two adjacent trimeric globular domains and a single stalk composed of collagen domains from two trimers. Although not necessary for trimer formation or stability, two of the three monomers in an Acrp30 trimer are covalently linked by a disulfide bond between cysteine residues at position 22. In contrast, assembly of hexameric and higher molecular weight (HMW) forms of Acrp30 depends upon formation of Cys22-mediated disulfide bonds because their reduction with dithiothreitol or substitution of Cys22 with alanine led exclusively to trimers. HMW and hexamer isoforms of Acrp30 activated NF-kappaB in C2C12 cells, but trimers, either natural, formed by reduction of Acrp30 hexamer, or formed by the C22A mutant, did not. In contrast, incubation of isolated rat extensor digitorum longus with naturally formed Acrp30 trimers or trimeric C22A Acrp30 led to increased phosphorylation of AMP-activated protein kinase-alpha at Thr172 and its activation. Hexameric and HMW Acrp30 could not activate AMP-activated

protein kinase. Thus, trimeric and HMW/hexameric Acrp30 activate different signal transduction pathways, and Acrp30 represents a novel example of the control of ligand signaling via changes in its oligomerization state.

3/7/47 (Item 47 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

17609575 BIOSIS NO.: 200300568294
Impaired multimerization of human adiponectin mutants associated with diabetes. Molecular structure and multimer formation of adiponectin.
AUTHOR: Waki Hironori; Yamauchi Toshimasa; Kamon Junji; Ito Yusuke; Uchida Shoko; Kita Shunbun; Hara Kazuo; Hada Yusuke; Vasseur Francis; Froguel Philippe; Kimura Satoshi; Nagai Ryozo; Kadowaki Takashi (Reprint)
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JOURNAL: Journal of Biological Chemistry 278 (41): p40352-40363 October 10, 2003 2003
MEDIUM: print
ISSN: 0021-9258
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: Adiponectin is an adipocyte-derived hormone, which has been shown to play important roles in the regulation of glucose and lipid metabolism. Eight mutations in human adiponectin have been reported, some of which were significantly related to diabetes and hypoadiponectinemia, but the molecular mechanisms of decreased plasma levels and impaired action of adiponectin mutants were not clarified. Adiponectin structurally belongs to the complement 1q family and is known to form a characteristic homomultimer. Herein, we demonstrated that simple SDS-PAGE under non-reducing and non-heat-denaturing conditions clearly separates multimer species of adiponectin. Adiponectin in human or mouse serum and adiponectin expressed in NIH-3T3 or Escherichia coli formed a wide range of multimers from trimers to high molecular weight (HMW) multimers. A disulfide bond through an amino-terminal cysteine was required for the

formation of multimers larger than a trimer. An amino-terminal Cys-Ser
mutation, which could not form multimers larger than a trimer, abrogated
the effect of adiponectin on the AMP-activated protein kinase
pathway in hepatocytes. Among human adiponectin mutations, G84R and
G90S mutants, which are associated with diabetes and hypoadiponectinemia,
did not form HMW multimers. R112C and I164T mutants, which are associated
with hypoadiponectinemia, did not assemble into trimers, resulting in
impaired secretion from the cell. These data suggested impaired
multimerization and/or the consequent impaired secretion to be among the
causes of a diabetic phenotype or hypoadiponectinemia in subjects having
these mutations. In conclusion, not only total concentrations, but also
multimer distribution should always be considered in the
interpretation of plasma adiponectin levels in health as well as
various disease states.

3/7/48 (Item 48 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

17503204 BIOSIS NO.: 200300458815
Impaired multimerization of human adiponectin mutants associated with diabetes.
AUTHOR: Waki Hironori (Reprint); Yamauchi Toshimasa (Reprint); Kamon Junji
(Reprint); Ito Yusuke (Reprint); Uchida Shoko (Reprint); Kita Shunbun
(Reprint); Hara Kazuo (Reprint); Hada Yusuke (Reprint); Kimura Satoshi
(Reprint); Nagai Ryozo (Reprint); Kadowaki Takashi (Reprint)
AUTHOR ADDRESS: Tokyo, Japan**Japan
JOURNAL: Diabetes 52 (Supplement 1): pA1 2003 2003
MEDIUM: print
CONFERENCE/MEETING: 63rd Scientific Sessions of the American Diabetes Association New Orleans, LA, USA June 13-17, 2003; 20030613
SPONSOR: American Diabetes Association
ISSN: 0012-1797 _(ISSN print)
DOCUMENT TYPE: Meeting; Meeting Abstract
RECORD TYPE: Citation
LANGUAGE: English

3/7/49 (Item 49 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

17379651 BIOSIS NO.: 200300336394
C1q-TNF Related Protein-1 (CTRP1) Prevents Thrombus Formation in Non-Human
Primates and Atherosclerotic Rabbits without Causing Bleeding.
AUTHOR: Meehan Woerner P (Reprint); Knitter Glenn H (Reprint); Lasser Gerald W (Reprint); Lewis Ken (Reprint); Ulla Marzec M (Reprint); Bishop
Paul D (Reprint); Hanson Stephen R (Reprint); Fruebis Joachim (Reprint)
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JOURNAL: Blood 100 (11): pAbstract No. 75 November 16, 2002 2002
MEDIUM: print
CONFERENCE/MEETING: 44th Annual Meeting of the American Society of Hematology Philadelphia, PA, USA December 06-10, 2002; 20021206
SPONSOR: American Society of Hematology
ISSN: 0006-4971
DOCUMENT TYPE: Meeting; Meeting Abstract; Meeting Poster
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: Injury to the arterial wall, often caused by rupturing atherosclerotic plaques, exposes pro-thrombotic agents including collagen
to circulating blood. As a consequence, circulating platelets adhere to
the site of injury and become fully activated, ultimately leading to thrombus formation that can trigger heart attack or stroke. The most effective anti-thrombotic therapies are designed to inhibit platelet function, possibly putting the patient at risk for severe bleeding complications. A less well-studied approach is to mask the binding sites
on collagen thereby inhibiting interaction with platelets. We discovered
a protein expressed in the vascular wall, CTRP1, which has a high binding
affinity for collagen. CTRP1 is a member of the C1q-TNF Related Protein
family that also includes C1q and Adiponectin. We hypothesized that CTRP1 could prevent platelet activation and thrombus formation by binding
to collagen. In vitro, CTRP1 inhibited platelet binding to collagen and
prevented platelet activation and aggregation dose-dependently. In vivo, CTRP1 was tested using a baboon arteriovenous (AV) shunt model and
a Folts vascular injury model. The AV-shunt model follows the accumulation of 111In-labeled platelets on a collagen-coated graft. The

grafts were pretreated with CTRP1 or saline and then placed in the AV
circuit for 60 minutes. Treatment with CTRP1 led to significantly reduced
platelet accumulation (1.86+-1.00X109 vs. 0.28+-0.32X109 platelets bound
in 6 control and 5 treated animals respectively; p=0.006). Using the
Folts model of cyclic flow variation in rabbits and cynomolgus monkeys,
we demonstrated that treatment with CTRP1 abolished cyclic flow
variations and maintained patency of injured, stenosed vessels. A further
refinement of the Folts vascular injury model was achieved using rabbits
with atherosclerotic lesions. Animals were placed on a high cholesterol
diet for two weeks and then underwent balloon denudation of the right
iliac artery. Three weeks after balloon injury, extensive atherosclerotic
lesions covered the denuded region. A crush injury was made rupturing the
atherosclerotic plaque and a stenosis was positioned over the injured
segment. Once cyclic flow was established, animals were treated with 1.0
mg/kg CTRP1 or BSA. Treatment with CTRP1 resulted in reduction of
platelet aggregation, increased blood flow, and maintained vessel
patency. Unlike systemically acting platelet antagonists, CTRP1 had no
effect on bleeding at therapeutic doses. This was demonstrated by
following parameters of hemostasis as well as by performing template
bleeds. In conclusion, CTRP1 is a potent anti-thrombotic protein that
acts locally at the site of vascular injury by binding to collagen.
Unlike conventional, systemically acting platelet antagonists it does not
produce increased bleeding or a decrease in circulating platelet numbers.

3/7/50 (Item 50 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

17289432 BIOSIS NO.: 200300248151
Adiponectin is markedly increased in patients with nephrotic syndrome
and is related to metabolic risk factors.
AUTHOR: Zoccali Carmine (Reprint); Mallamaci Francesca; Panuccio Vincenzo;
Tripepi Giovanni; Cutrupi Sebastiano; Parlongo Saverio; Catalano
Francesco; Tanaka Sachiyo; Ouchi Noriyuki; Kihara Shinji; Funahashi Tohru

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JOURNAL: Kidney International 63 (Supplement 84): pS98-S102 May 2003 2003
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RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: Background. Adiponectin (ADPN), the gene product of apM1, is the most abundant secretory protein of the adipose tissue in human plasma. Altered regulation (reduced synthesis) of this substance may be relevant to endothelial dysfunction and cardiovascular complications in patients with ESRD. Methods. We investigated the relationship between plasma ADPN, glomerular filtration rate (GFR) (plasma iohexol clearance), and metabolic risk factors in 16 patients with nephrotic syndrome, in 25 patients with chronic nephropathies without nephrotic syndrome, and in 31 healthy subjects. Results. Plasma ADPN was much higher ($P < 0.01$) in patients with nephrotic syndrome (24.4 +- 14.9 mug/mL) than in patients with chronic nephropathies without nephrotic syndrome (12.3 +- 7.2 mug/mL) and healthy subjects (5.9 +- 2.6 mug/mL). In the aggregate 24-hour, proteinuria ($r = 0.53$, $P < 0.01$) and serum cholesterol ($r = 0.53$, $P < 0.01$) were strong and direct correlates of plasma ADPN, while serum albumin correlated inversely ($r = -0.46$, $P < 0.01$) with this protein. Proteinuria appeared to be an important confounder of the relationship between ADPN and the GFR because in the whole patient population (with and without nephrotic syndrome), this relationship emerged only after data adjustment for 24-hour proteinuria (partial $r = -0.31$, $P = 0.05$), while no such relationship was demonstrable on crude data analysis ($r = 0.03$, $P = 0.87$). Conclusions. ADPN is markedly increased in patients with nephrotic syndrome, and proteinuria is strongly related to circulating ADPN in patients with nephrotic and non-nephrotic renal diseases. The relationships between plasma ADPN, serum cholesterol, and serum albumin suggest that this adipocyte protein may serve to mitigate endothelial damage triggered by dyslipidemia and other risk factors in patients with chronic renal diseases.

3/7/51 (Item 1 from file: 24)
DIALOG(R)File 24:CSA Life Sciences Abstracts

0003622049 IP ACCESSION NO: 8907684
Globular adiponectin increases cGMP formation in blood platelets independently of nitric oxide

RIBA, R; PATEL, B; ABURIMA, A; NASEEM, KM
Centre for Atherothrombosis Research, Medical Biosciences, University of
Bradford, Bradford, UK, [mailto:k.m.naseem@bradford.ac.uk]

Journal of Thrombosis and Haemostasis, Suppl. 12, v 6, p 2121-2131, December 2008
PUBLICATION DATE: 2008

PUBLISHER: Blackwell Publishing Ltd., 9600 Garsington Road

DOCUMENT TYPE: Journal Article
RECORD TYPE: Abstract
LANGUAGE: English
SUMMARY LANGUAGE: English
ISSN: 1538-7933
ELECTRONIC ISSN: 1538-7836
DOI: 10.1111/j.1538-7836.2008.03179.x
FILE SEGMENT: Calcium & Calcified Tissue Abstracts

ABSTRACT:
Summary.Background:Platelet-derived nitric oxide (NO) has been shown to play conflicting roles in platelet function, although it is accepted that NO mediates its actions through soluble guanylyl cyclase (sGC). This confusion concerning the roles of platelet NO may have arisen because of an uncharacterized mechanism for activation of sGC. Objectives:To examine the ability of the novel platelet agonist globular adiponectin (gAd) to stimulate the NO-independent cGMP-protein kinase G (PKG) signaling cascade. Methods:We used three independent markers of NO signaling, [3H]l-citrulline production, cGMP accrual, and immunoblotting of vasodilator-stimulated phosphoprotein (VASP), to examine the NO signaling cascade in response to gAd. Results:gAd increased platelet cGMP formation, resulting in a dose- and time-dependent increase in phospho-VASP157/239. Phosphorylation of VASP

in response to gAd was mediated by both protein kinase A and PKG. Importantly, cGMP formation occurred in the absence of NO synthase (NOS) activation and in the presence of NOS inhibitors. Indeed, inhibition of the NOS signaling cascade had no influence on gAd-mediated platelet aggregation. Exploration of the mechanism demonstrated that NO-independent cGMP formation, phosphorylation of VASP and association of sGC alpha 1 with heat shock protein-90 induced by gAd were blocked under conditions that inhibited Src kinases, implying a tyrosine kinase-dependent mechanism. Indeed, sGC alpha 1 was reversibly tyrosine phosphorylated in response to gAd, collagen, and collagen-related peptide, an effect that required Src kinases and downstream Ca2+ mobilization. Conclusions:These data demonstrate activation of the platelet cGMP signaling cascade by a novel tyrosine kinase-dependent mechanism in the absence of NO.

3/7/52 (Item 2 from file: 24)
DIALOG(R)File 24:CSA Life Sciences Abstracts

0002989892 IP ACCESSION NO: 7205032
Increased hypothalamic 5-HT2A receptor gene expression and effects of pharmacologic 5-HT2A receptor inactivation in obese A super(y) mice

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Biochemical and Biophysical Research Communications, v 351, n 4, p 1078-1082, December 29, 2006
PUBLICATION DATE: 2006

PUBLISHER: Elsevier Science B.V., P.O. Box 211 Amsterdam 1000 AE Netherlands, [mailto:nlinfo-f@elsevier.nl], [URL:http://www.elsevier.nl/]

DOCUMENT TYPE: Journal Article
RECORD TYPE: Abstract
LANGUAGE: English
SUMMARY LANGUAGE: English
ISSN: 0006-291X
DOI: 10.1016/j.bbrc.2006.10.173

FILE SEGMENT: CSA Neurosciences Abstracts

ABSTRACT:
Serotonin (5-hydroxytryptamine; 5-HT) 2A receptors contribute to the effects of 5-HT on platelet aggregation and vascular smooth muscle cell proliferation, and are reportedly involved in decreases in plasma levels of adiponectin, an adipokine, in diabetic subjects. Here, we report that systemic administration of sarpogrelate, a 5-HT2A receptor antagonist, suppressed appetite and increased hypothalamic pro-opiomelanocortin and cocaine- and amphetamine-regulated transcript,
corticotropin releasing hormone, 5-HT2C, and 5-HT1B receptor gene expression. A super(y) mice, which have ectopic expression of the agouti
protein, significantly increased hypothalamic 5-HT2A receptor gene expression in association with obesity compared with wild-type mice matched
for age. Systemic administration of sarpogrelate suppressed overfeeding,
body weight gain, and hyperglycemia in obese A super(y) mice, whereas it
did not increase plasma adiponectin levels. These results suggest that obesity increases hypothalamic 5-HT2A receptor gene expression, and
pharmacologic inactivation of 5-HT2A receptors inhibits overfeeding and
obesity in A super(y) mice, but did not increase plasma adiponectin levels.

3/7/53 (Item 1 from file: 34)
DIALOG(R)File 34:SciSearch(R) Cited Ref Sci

18880688 Genuine Article#: 408KA Number of References: 22
Title: Altered Molecular Weight Forms of Adiponectin in Hypertension
Author(s): Baumann M (REPRINT) ; von Eynatten M; Dan L; Richart T; Kouznetsova T; Heemann U; Staessen JA
Corporate Source: Tech Univ Munich,Klinikum Rechts Isar, Dept Nephrol,Ismaninger Str 22/D-81675 Munich//Germany/ (REPRINT); Tech Univ Munich,Klinikum Rechts Isar, Dept Nephrol,D-81675 Munich//Germany/; Maastricht Univ,Dept Epidemiol, Genet Epidemiol Unit,Maastricht//Netherlands/; Katholieke Univ Leuven,Dept Cardiovasc Dis, Div Hypertens & Cardiac Rehabil, Studies Coordinating Ctr,Louvain//Belgium/
Journal: JOURNAL OF CLINICAL HYPERTENSION, 2009, V11, N1 (JAN), P11-16
ISSN: 1524-6175 Publication date: 20090100
Publisher: WILEY-BLACKWELL PUBLISHING, INC, COMMERCE PLACE, 350 MAIN ST,

MALDEN 02148, MA USA
Language: English Document Type: ARTICLE
Abstract: An important link between adiponectin and hypertension has been proposed in clinical studies. In the circulation, adiponectin is predominantly present in multimeric complexes, of which high-molecular weight (HMW) adiponectin is thought to represent the biological active form. The authors investigated which role the different multimeric adiponectin isoforms play in context with hypertension as compared to total adiponectin levels. Fifty (19 normotensive /31 hypertensive) patients were included in the study. Total adiponectin and adiponectin multimers were determined by enzyme-linked immunosorbent assay and western blot. The authors analyzed associations between adiponectin multimer levels and blood pressure. Total adiponectin concentrations were not significantly different between hypertensive and normotensive patients (6.8 +/- 2.3 vs 7.5 +/- 4.2 mu g/mL). HMW adiponectin was significantly lower (P<.05) and low-molecular weight adiponectin was significantly higher (P<.01) in hypertensive than in normotensive persons (3.8 +/- 1.7 vs 5.2 +/- 3.0 mu g/mL and 0.9 +/- 0.5 vs 1.8 +/- 0.9, respectively). Low molecular weight was an independent predictor for the presence of hypertension (effect coefficient: 0.160-0.445; P<.001) in multivariate analyses. These results suggest that the composition of the molecular weight forms of adiponectin in hypertension are characterized by reduced HMW adiponectin, the proposed major active form of adiponectin, and increased low-molecular weight adiponectin . Moreover, the latter represents an independent predictor of prevalent hypertension, suggesting an association between adiponectin multimer composition and hypertension. J Clin Hypertens (Greenwich). 2009; 11: 11-16. (C) 2009 Wiley Periodicals, Inc.

3/7/54 (Item 2 from file: 34)
DIALOG(R)File 34:SciSearch(R) Cited Ref Sci

18804396 Genuine Article#: 400CF Number of References: 43
Title: Adipokines and Acute Coronary Syndrome
Author(s): Nakas-Icindic E (REPRINT) ; Valjevac A; Zaciragic A
Corporate Source: Univ Sarajevo,Fac Med, Inst Physiol & Biochem,Sarajevo 71000//Bosnia & Herceg/ (REPRINT); Univ Sarajevo,Fac Med, Inst Physiol

& Biochem,Sarajevo 71000//Bosnia & Herceg/
Journal: HEALTHMED, 2008, V2, N4, P225-233
ISSN: 1840-2291 Publication date: 20080000
Publisher: DRUNPP-SARAJEVO, BOLNICKA BB, SARAJEVO, 71000, BOSNIA & HERCEG
Language: English Document Type: ARTICLE
Abstract: Adipose tissue has traditionally been considered as a tissue devoted mainly to energy storage. Now it is recognized as a multifunctional organ involved in the production of hormones, growth factors and cytokines named adipokines.

In obese subject the production of adipokines is impaired, In obesity high level of leptin, resistin, and low level of adiponectin have been observed and implicated in insulin resistance, atherosclerosis and metabolic syndrome.

In patients with established coronary atherosclerosis increased body weight is an independent predictor of ail acute coronary syndrome. The exact mechanism of obesity induced coronary heart disease is not fully elucidated. Current research is aimed to determine links between adipokines and coronary heart disease.

Leptin, adiponectin and resistin are adipokines that are implicated in coronary endothelial dysfunction, trombogenesis and inflammation. These processes are known to precipitate atherosclerotic plaque rupture and acute coronary syndrome.

Recent studies demonstrated that high plasma leptin and low adiponectin levels as observed in obese subjects impair coronary acetylcholine-mediated vasodilatation in vitro and in vivo. Resistin also impair coronary vasodilatation but via bradykinin pathway.

Leptin and resistin show proinflammatory effects upregulating cytokine production in macrophages and might lead to destabilization of coronary atherosclerotic plaque. Leptin has been observed 10 Stimulate angiogenesis, platelet aggregation, and atherothrombosis in obese human. In obese subject the production of adiponectin, which has protective effects oil coronary blood vessels is suppressed.

This paper summarizes the role of three adipokines: leptin, resistin and adiponectin in acute coronary syndrome and implicates theirs possible appliance in clinical practice.

3/7/55 (Item 3 from file: 34)
DIALOG(R)File 34:SciSearch(R) Cited Ref Sci

18720900 Genuine Article#: 393ML Number of References: 43
Title: Leptin, but not adiponectin, is a predictor of recurrent cardiovascular events in men: results from the LIPID study
Author(s): Soderberg S (REPRINT) ; Colquhoun D; Keech A; Yallop J; Barnes EH; Pollicino C; Simes J; Tonkin AM; Nestel P
Corporate Author(s): LIPID Study Investigators
Corporate Source: Umea Univ Hosp,Dept Publ Hlth & Clin Med,SE-90185 Umea//Sweden/ (REPRINT); Umea Univ Hosp,Dept Publ Hlth & Clin Med,SE-90185 Umea//Sweden/; Univ Queensland,Brisbane/Qld/Australia/; Univ Sydney,Natl Hlth & Med Res Council Clin Trials Ctr,Sydney/NSW 2006/Australia/; Monash Univ,Dept Epidemiol & Prevent Med,Melbourne/Vic 3004/Australia/; Natl Heart Fdn Australia,Melbourne/Vic/Australia/; Baker Heart Res Inst,Melbourne/Vic/Australia/
Journal: INTERNATIONAL JOURNAL OF OBESITY, 2009, V33, N1 (JAN), P123-130
ISSN: 0307-0565 Publication date: 20090100
Publisher: NATURE PUBLISHING GROUP, MACMILLAN BUILDING, 4 CRINAN ST, LONDON N1 9XW, ENGLAND
Language: English Document Type: ARTICLE
Abstract: Objective: To investigate the relationships between plasma leptin and adiponectin levels and recurrent cardiovascular events (cardiovascular death, nonfatal myocardial infarction and stroke) in men with earlier acute coronary syndromes.

Design, subjects and measurements: A nested case-control study examined circulating leptin and adiponectin levels in plasma obtained 4-6 years after entry into the Long-Term Intervention with Pravastatin in Ischaemic Disease (LIPID) trial. Plasma was assayed from 184 men who suffered recurrent events within 4.4 years after blood collection and 184 matched controls who remained free of further events. The association between cardiovascular events and the explanatory variables was examined by conditional logistic regression analysis.

Results: Relative risk (RR) increased across increasing leptin quartiles; the highest quartile compared with the lowest quartile was related to the highest risk (P for trend = 0.002); the increased risk

remained after adjustment for risk factors (P = 0.018) or for obesity
(P = 0.038), but in the final model (adjusted for randomized treatment,
other drugs, LIPID risk score, age and body mass index), the risk was
attenuated (RR = 1.61, 95% CI: 0.72-3.57, P for trend = 0.34).
Adiponectin did not predict cardiovascular events. Subjects
randomly allocated to pravastatin had 6% lower leptin levels (P = 0.04)
than those allocated to placebo.
Conclusion: Plasma leptin was a significant and independent
predictor of recurrent cardiovascular events (cardiovascular death,
nonfatal myocardial infarction and stroke) in men with earlier acute
coronary syndromes.

3/7/56 (Item 4 from file: 34)
DIALOG(R)File 34:SciSearch(R) Cited Ref Sci

18594513 Genuine Article#: 379OJ Number of References: 44
Title: Adiponectin Multimers and Metabolic Syndrome Traits: Relative Adiponectin Resistance in African Americans
Author(s): Lara-Castro C (REPRINT) ; Doud EC; Tapia PC; Munoz AJ; Fernandez
JR; Hunter GR; Gower BA; Garvey WT
Corporate Source: Univ Alabama,Dept Nutr Sci,Birmingham//AL/35294 (REPRINT)
; Univ Alabama,Dept Nutr Sci,Birmingham//AL/35294; Birmingham Vet Affairs Med Ctr,Birmingham//AL/
Journal: OBESITY, 2008, V16, N12 (DEC), P2616-2623
ISSN: 1930-7381 Publication date: 20081200
Publisher: NATURE PUBLISHING GROUP, 75 VARICK STREET, 9TH FLOOR, NEW YORK,
NY 10013-1917 USA
Language: English Document Type: ARTICLE
Abstract: African Americans (AAs) tend to have lower total adiponectin levels compared to European Americans (EA); however, it is not known whether race affects adiponectin multimer distribution and their relationships to metabolic traits. We measured
total adiponectin, high molecular weight (HMW), low molecular weight (LMW) (i.e., hexamer), and trimer adiponectin in 132 normoglycemic premenopausal women (75 AAs, 57 EAs), together with measures of total and abdominal fat, plasma lipids, insulin sensitivity
(S-i), and genetic admixture estimates. We found that lower total adiponectin in AAs was explained by reduced LMW, and trimer forms because levels of HMW did not differ between races. In EAs, HMW was

highly correlated with multiple metabolic syndrome traits. In contrast,
the LMW and trimer forms were most highly correlated with metabolic
traits in AAs, including abdominal adiposity, lipids, and S-i. At
similar levels of visceral adiposity, AAs exhibited significantly lower
LMW adiponectin than EAs. Similarly, at comparable levels of HMW
and LMW adiponectin, AAs were more insulin resistant than their
EA counterparts. In conclusion, (i) serum adiponectin is lower in
AAs predominantly as a result of reduced concentrations of LMW and
trimers multimeric forms; (ii) LMW and trimer, not HMW, are most
broadly correlated with metabolic traits in AAs. Thus, HMW
adiponectin may exert less bioactivity in explaining the
metabolic syndrome trait cluster in populations of predominant African
genetic background.

3/7/57 (Item 5 from file: 34)
DIALOG(R)File 34:SciSearch(R) Cited Ref Sci

18545398 Genuine Article#: 376GU Number of References: 268
Title: Adiponectin multimers in maternal plasma
Author(s): Mazaki-Tovi S; Romero R (REPRINT) ; Kusanovic JP; Erez O;
Vaisbuch E; Gotsch F; Mittal P; Than GN; Nhan-Chang C; Chaiworapongsa T
; Edwin S; Camacho N; Nien JK; Hassan SS
Corporate Source: Hutzel Womens Hosp,Perinatol Res Branch, Intramural Div,
NICHD,NIH,DHHS,Box 4,3990 John R/Detroit//MI/48201 (REPRINT); Hutzel
Womens Hosp,Perinatol Res Branch, Intramural Div,
NICHD,NIH,DHHS,Detroit//MI/48201; Hutzel Womens Hosp,Perinatol Res
Branch, Intramural Div, NICHD,NIH,DHHS,Bethesda//MD/; Wayne State
Univ,Hutzel Womens Hosp, Dept Obstet & Gynecol,Detroit//MI/; Wayne
State Univ,Ctr Mol Med & Genet,Detroit//MI/; Univ Catolica Chile,Hosp
Sotero Rio, Ctr Perinatal Diag & Res CEDIP,Puente Alto//Chile/
Journal: JOURNAL OF MATERNAL-FETAL & NEONATAL MEDICINE, 2008, V21, N11, P
796-815
ISSN: 1476-7058 Publication date: 20080000
Publisher: TAYLOR & FRANCIS LTD, 4 PARK SQUARE, MILTON PARK, ABINGDON OX14
4RN, OXON, ENGLAND
Language: English Document Type: REVIEW
Abstract: Objective. Adiponectin is an anti-diabetic,
anti-atherogenic, anti-inflammatory, and angiogenic adipokine that
circulates in oligomeric complexes including: low molecular weight
(LMW) trimers, medium molecular weight (MMW) hexamers, and high

molecular weight (HMW) isoforms. The aim of this study was to determine whether there are changes in adiponectin multimers in pregnancy and as a function of maternal weight. Study design. In this cross-sectional study, plasma concentrations of total, HMW, MMW, and LMW adiponectin were determined in women included in three groups: (1) normal pregnant women of normal body mass index (BMI) (n=466), (2) overweight pregnant women (BMI 25; n=257), and (3) non-pregnant women of normal weight (n=40). Blood samples were collected once from each woman between 11 and 42 weeks of gestation. Plasma adiponectin multimer concentrations were determined by enzyme-linked immunosorbent assay (ELISA). Non-parametric statistics were used for analysis. Results. (1) The median HMW adiponectin concentration and the median HMW/total adiponectin ratio were significantly higher, and the median LMW adiponectin concentration was significantly lower in pregnant women than in non-pregnant women. (2) Among pregnant women, the median plasma concentration of total, HMW, and MMW adiponectin was significantly higher in normal weight women than in overweight patients. (3) Maternal HMW was the most prevalent adiponectin multimer regardless of gestational age or BMI status. (4) There were no significant differences in the median concentration of total, MMW, and LMW adiponectin and their relative distribution with advancing gestation. Conclusion. Human pregnancy is characterized by quantitative and qualitative changes in adiponectin multimers, especially the most active isoform, HMW adiponectin.

3/7/58 (Item 6 from file: 34)
DIALOG(R)File 34:SciSearch(R) Cited Ref Sci

18234265 Genuine Article#: 343IK Number of References: 26
Title: Blockade of serotonin 2A receptor improves glomerular endothelial function in rats with streptozotocin-induced diabetic nephropathy
Author(s): Kobayashi S; Satoh M (REPRINT) ; Namikoshi T; Haruna Y; Fujimoto S; Arakawa S; Komai N; Tomita N; Sasaki T; Kashihara N
Corporate Source: Kawasaki Med Univ,Dept Internal Med, Div Nephrol,577 Matsushima/Kurashiki/Okayama 7010192/Japan/ (REPRINT); Kawasaki Med Univ,Dept Internal Med, Div Nephrol,Kurashiki/Okayama 7010192/Japan/
Journal: CLINICAL AND EXPERIMENTAL NEPHROLOGY, 2008, V12, N2 (APR), P 119-125
ISSN: 1342-1751 Publication date: 20080400

Publisher: SPRINGER, 233 SPRING ST, NEW YORK, NY 10013 USA
Language: English Document Type: ARTICLE
Abstract: Background Serotonin (5-HT) is involved in vascular inflammation
and atherosclerogenesis. Serum 5-HT concentrations are elevated in diabetes, and 5-HT is involved in diabetic vasculopathies. Sarpogrelate
hydrochloride, a 5-HT2A receptor antagonist, has renoprotective effects, but its effect in diabetic nephropathy is not elucidated. The
aim of this study was to examine the effects of sarpogrelate on endothelial dysfunction in rats with streptozotocin (STZ)induced diabetes.

Methods Rats with STZ-induced diabetes were either untreated or
treated with sarpogrelate (30 mg/kg P.O.) for 8 weeks. At the end of
the experiment, we measured urinary albumin excretion, serum adiponectin concentration and platelet-derived microparticles. Intraglomerular coagulation was detected by immunostaining for platelets. Production of renal reactive oxygen species (ROS) and nitric
oxide (NO) was investigated by confocal laser microscopy and used as an
index of glomerular endothelial dysfunction.

Results Diabetic nephropathy was associated with enhanced production of ROS and diminished bioavailable NO in the glomeruli. Treatment with sarpogrelate improved ROS/NO imbalance in glomeruli,
suppressed platelet aggregation in glomeruli, reduced platelet-derived microparticles, increased serum adiponectin level and reduced the level of albuminuria, compared with nontreated
diabetic rats.

Conclusions Our results indicate that sarpogrelate improves endothelial function in rats with STZ-induced diabetes through a reduction of glomerular platelet activation and an increase in serum
adiponectin concentrations and suggest that sarpogrelate is potentially useful for the treatment of diabetic nephropathy.

3/7/59 (Item 7 from file: 34)
DIALOG(R)File 34:SciSearch(R) Cited Ref Sci

17940703 Genuine Article#: 309IS Number of References: 34
Title: Adiponectin induces dentin sialophosphoprotein in rat dental pulp cells: An in vitro study
Author(s): Yasuda Y (REPRINT) ; Koike T; Kawamorita T; Saito T

Corporate Source: Hlth Sci Univ Hokkaido,Sch Dent, Dept Oral Rehabil, Div
Clin Cariol & Endodontol,1757 Kanazawa/Ishikari/Hokkaido 0610293/Japan/
(REPRINT); Hlth Sci Univ Hokkaido,Sch Dent, Dept Oral Rehabil, Div Clin
Cariol & Endodontol,Hokkaido//Japan/
Journal: JOURNAL OF ENDODONTICS, 2008, V34, N6 (JUN), P679-683
ISSN: 0099-2399 Publication date: 20080600
Publisher: ELSEVIER SCIENCE INC, 360 PARK AVE SOUTH, NEW YORK, NY 10010-1710 USA
Language: English Document Type: ARTICLE
Abstract: Adiponectin is known to play an important role in the regulation of blood glucose levels through the mediation of adiponectin receptors 1 and 2 (AR1 and AR2, respectively). The purpose of this study was to investigate the role of adiponectin in dental pulp cells. The expressions of both AR1 and AR2 were observed in dental pulp by reverse transcriptase polymerase chain reaction (RT-PCR) and Western blotting. Quantitative analysis of Alizarin Red S staining showed that 10 mu g/mL of adiponectin significantly promoted mineralization by 1.6 times compared with control on day 12. However, no significant difference in mineralization was observed between control and 0.1 or 1 mu g/mL adiponectin treatment. Moreover, real-time PCR results indicated that adiponectin (10 mu g/mL) significantly increased the expression of dentin sialophosphoprotein (DSPP) by 2.3 and 1.8 times compared with control on days 8 and 12, respectively. These results indicated that adiponectin might promote mineralization by inducing DSPP expression in dental pulp cells.

3/7/60 (Item 8 from file: 34)
DIALOG(R)File 34:SciSearch(R) Cited Ref Sci

17846964 Genuine Article#: 302BQ Number of References: 30
Title: Globular adiponectin induces platelet activation through the collagen receptor GPVI-Fc receptor gamma chain complex
Author(s): Riba R; Hughes CE; Graham A; Watson SP; Naseem KM (REPRINT)
Corporate Source: Univ Bradford,Ctr Atherothrombosis Res,Bradford BD7 1DP/W
Yorkshire/England/ (REPRINT); Univ Bradford,Ctr Atherothrombosis Res,Bradford BD7 1DP/W Yorkshire/England/; Inst Biomed Res,Ctr Cardiovasc Sci,Birmingham//AL/; Glasgow Caledonian Univ,Vascular Biol Grp Biol & Biomed Sci,Glasgow G4 0BA/Lanark/Scotland/
Journal: JOURNAL OF THROMBOSIS AND HAEMOSTASIS, 2008, V6, N6 (JUN), P

1012-1020
ISSN: 1538-7933 Publication date: 20080600
Publisher: BLACKWELL PUBLISHING, 9600 GARSINGTON RD, OXFORD OX4 2DQ, OXON,
ENGLAND
Language: English Document Type: ARTICLE
Abstract: Background: The adipocyte-derived cytokine, adiponectin (Ad), exerts potent vascular effects, although the direct effects of Ad on blood platelets are unclear. Objective: The influence of globular Ad (gAd) on blood platelet function was investigated. Research design and methods: We measured platelet aggregation and tyrosine phosphorylation signaling events in human and mouse platelets. The ability of gAd to activate Glycoprotein VI (GPVI) activity was determined with a NFAT luciferase reporter assay. Results: gAd, but not full length Ad, induced rapid aggregation and granule secretion of human and mouse platelets through a pathway that is ablated under conditions of Src kinase inhibition, indicating a tyrosine kinase-dependent mechanism. Consistent with this, gAd stimulates rapid tyrosine phosphorylation of several proteins in human and mouse platelets. The pattern of increase in tyrosine phosphorylation was similar to that induced by collagen, with the tyrosine kinase Syk and PLC gamma 2 being identified among the list of tyrosine phosphorylated proteins. As collagen activates platelet through the GPVI-Fc receptor gamma-chain (FcR gamma) complex, we used FcR gamma null platelets (which also lack GPVI) to explore the mechanism by which gAd stimulates platelets. Stimulation of tyrosine phosphorylation and platelet aggregation by gAd was abolished in FcR gamma null platelets and markedly reduced in the absence of PLC gamma 2. Further, GPVI was confirmed as a collagen receptor for gAd by increased luciferase activity in Jurkat T-cells transfected with GPVI. Conclusions: We identify gAd as a novel ligand for GPVI that stimulates tyrosine kinase-dependent platelet aggregation. Our data raise the possibility that gAd may promote unwanted platelet activation at sites of vascular injury.

3/7/61 (Item 9 from file: 34)
DIALOG(R)File 34:SciSearch(R) Cited Ref Sci

17679944 Genuine Article#: 283HX Number of References: 49

Title: Adiponectin: An adipocytic hormone implicated in carbohydrate homeostasis and cardiovascular fonction
Author(s): Guerre-Millo M (REPRINT)
Corporate Source: Hop Hotel Dieu,AP HP, INSERM, Serv Nutr,U 755,1 Pl Parvis Notre Dame/F-75004 Paris//France/ (REPRINT); Hop Hotel Dieu,AP HP, INSERM, Serv Nutr,U 755,F-75004 Paris//France/
Journal: SANG THROMBOSE VAISSEAUX, 2007, V19, N5 (MAY), P255-260
ISSN: 0999-7385 Publication date: 20070500
Publisher: JOHN LIBBEY EUROTEXT LTD, 127 AVE DE LA REPUBLIQUE, 92120 MONTROUGE, FRANCE
Language: French Document Type: REVIEW
Abstract: The discovery of leptin and adiponectin, two proteins produced by adipose cells and released in the circulation, is considered as a breakthrough in the field of metabolic diseases. Leptin has been clearly characterized as a hormone, which modulates food intake depending on energy status. By contrast, a biological role for adiponectin has not yet been firmly established. Nevertheless, numerous clinical and experimental observations indicate that low adiponectin plasma levels contribute to the pathogenesis of insulin resistance, type 2 diabetes and cardiovascular diseases in obese or overweight patients. Indeed, adiponectin exerts both antiatherogenic effects, by targeting vascular endothelial cells, and insulin-sensitizing effects, prominently in muscles and liver. Among several circulating forms, a high molecular weight multimer of adiponectin is thought to be the most clinically relevant. Adiponectin signalling pathways comprise at least two putative receptors, AdipoR1 and AdipoR2, which mediate increased fatty acid oxidation in muscles and fat, decreased glucose production in liver and reduced inflammation-related processes in endothelial cells, at least in part through activation of AMP kinase. Methods of improving adiponectin bioactivity, are under intense investigation. This includes the use of drugs such as thiazolidinediones and CB1 blockers (rimonabant), which increase adiponectin gene expression and plasma levels. Alternatively, the development of AdipoR agonists could prove beneficial in situations such as obesity, where decreased serum adiponectin levels are observed.

3/7/62 (Item 10 from file: 34)
DIALOG(R)File 34:SciSearch(R) Cited Ref Sci

17582579 Genuine Article#: 276LN Number of References: 40
Title: Genetic and environmental determinants of hepatocyte growth factor
levels and their association with obesity and blood pressure
Author(s): Vistoropsky Y; Trofimov S; Malkin I; Kobyliansky E; Livshits G
(REPRINT)
Corporate Source: Tel Aviv Univ,Sackler Fac Med, Dept Anat & Anthropol,Yoran Inst Human Genome Res, Human Populat Biol Res Unit,IL-69978 Tel Aviv//Israel/ (REPRINT); Tel Aviv Univ,Sackler Fac Med, Dept Anat & Anthropol,Yoran Inst Human Genome Res, Human Populat Biol Res Unit,IL-69978 Tel Aviv//Israel/
Journal: ANNALS OF HUMAN BIOLOGY, 2008, V35, N1 (JAN-FEB), P93-103
ISSN: 0301-4460 Publication date: 20080100
Publisher: TAYLOR & FRANCIS LTD, 4 PARK SQUARE, MILTON PARK, ABINGDON OX14 4RN, OXON, ENGLAND
Language: English Document Type: ARTICLE
Abstract: Background: Hepatocyte growth factor (HGF) is a member of the adipocytokine family; it is implicated in tissue repair, regeneration, and angiogenesis. Several studies have reported that the HGF plays important role in obesity and cardiovascular disease.

Aim: This study examines whether HGF and its phenotypic correlations with obesity and blood pressure (BP), in healthy individuals, are due to shared genetic or common environmental factors.

Subjects and methods: Body mass index (BMI), waist-to-hip ratio (WHR), BP, and HGF plasma concentrations were measured in a sample of 733 individuals belonging to 248 pedigrees.

Results: The most significant phenotypic correlations were found among HGF, WHR, and systolic BP ($p < 0.001$). Analysis of the familial aggregation revealed that parent-offspring and sibling correlations in HGF levels, adjusted for age, age 2, and sex, were statistically highly significant ($p < 0.001$). Variance decomposition analysis showed that when adjusted for potential covariates, 48.4% of the HGF variation was due to putative genetic factors. The genetic correlations between all pairs of studied traits (HGF, WHR, and SBP) were statistically significant ($p < 0.02$) and ranged between 0.23 +/-

0.07 and 0.40 +/- 0.07. However, correlation between WHR and BP becomes
non-significant after adjustment for HGF.

Conclusions: The results provide evidence that putative genetic
factors involved in regulation of HGF variation contribute also
significantly to variation of the obesity and BP. It is possible that
the familial resemblance for WHR and the SBP correlation in the studied
sample is affected substantially by genetic factors regulating
circulating HGF levels.

3/7/63 (Item 11 from file: 34)
DIALOG(R)File 34:SciSearch(R) Cited Ref Sci
(c) 2009 The Thomson Corp. All rts. reserv.

17440645 Genuine Article#: 256JY Number of References: 0
Title: 5-HT2 receptor blockade improves cerebral infarction in diabetic
rodent models by inducing adiponectin expression and inhibiting
platelet aggregation
Author(s): Nakagawa H; Uchida S; Yamada K; Shimada H; Akira T; Kitada Y
Corporate Source: Mitsubishi Pharma Corp,Yokohama/Kanagawa/Japan/
Journal: STROKE, 2008, V39, N2 (FEB), P658-658
ISSN: 0039-2499 Publication date: 20080200
Publisher: LIPPINCOTT WILLIAMS & WILKINS, 530 WALNUT ST, PHILADELPHIA, PA
19106-3621 USA
Language: English Document Type: MEETING ABSTRACT

3/7/64 (Item 12 from file: 34)
DIALOG(R)File 34:SciSearch(R) Cited Ref Sci
(c) 2009 The Thomson Corp. All rts. reserv.

17088144 Genuine Article#: 228OD Number of References: 31
Title: A low level of C-reactive protein in Japanese adults and its
association with cardiovascular risk factors: The Japan
NCVC-Collaborative Inflammation Cohort (JNIC) Study
Author(s): Saito I (REPRINT) ; Sato S; Nakamura M; Kokubo Y; Mannami T;
Adachi H; Konishi M; Okada K; Iso H; Kario K; Ohsuzu F; Momiyama Y;
Tsushima M
Corporate Source: Ehime Univ,Grad Sch Med, Dept Publ Hlth Social Med & Med
Informat,Toon/Ehime 7910295/Japan/ (REPRINT); Ehime Univ,Grad Sch Med,

Dept Publ Hlth Social Med & Med Informat,Toon/Ehime 7910295/Japan/;
Nara Med Univ,Dept Publ Hlth Policy,Kashihara/Nara 634/Japan/; Osaka
Med Ctr Hlth Sci & Promot,Osaka//Japan/; Natl Cardiovasc Ctr,Dept
Prevent Cardiol,Suita/Osaka 565/Japan/; Kagawa Univ,Fac Med, Dept Hyg
Publ Hlth,Kagawa//Japan/; Kurume Univ,Sch Med, Dept Internal Med, Div
Cardiovasc Med,Kurume/Fukuoka 830/Japan/; Osaka Univ,Grad Sch Med, Dept
Social & Environm Med,Suita/Osaka/Japan/; Jichi Med Sch,Dept
Cardiol,Tochigi//Japan/; Natl Def Med Coll,Dept Internal Med
1,Saitama//Japan/; Int Univ Hlth & Welfare,Atami Hosp, Dept Geriatr
Med,Atami//Japan/
Journal: ATHEROSCLEROSIS, 2007, V194, N1 (SEP), P238-244
ISSN: 0021-9150 Publication date: 20070900
Publisher: ELSEVIER IRELAND LTD, ELSEVIER HOUSE, BROOKVALE PLAZA, EAST PARK
SHANNON, CO, CLARE, 00000, IRELAND
Language: English Document Type: ARTICLE
Abstract: High-sensitivity C-reactive protein (hs-CRP) levels vary remarkably by race and ethnic group. We examined hs-CRP levels and their association with cardiovascular risk factors in the Japanese general population. The Japan National Cardiovascular Center (NCVC)-collaborative Inflammation Cohort (JNIC) Study recruited 5213
men and 7071 women aged >= 40 years from seven communities in Japan
during 2002-2004. hs-CRP was measured using nephelometry calibrated
with CRM 470, the international plasma protein reference material. Traditional cardiovascular risk factors and their aggregation
were studied in multivariate logistic models, stratified by overweight
status. Median hs-CRP levels in men and women were 0.60 and 0.45 mg/L,
respectively. The percentage of subjects with hs-CRP levels < 1.0, 1.0-3.0, and >3.0 mg/L was 67.4%, 22.0%, and 10.6% in men,
respectively, and 76.3%, 16.7%, and 7.0% in women. hs-CRP levels showed
significant linear associations with traditional risk factors. Overweight, hypertension, dyslipidemia (men only), smoking (men only),
and diabetes (women only) contributed significantly to elevated hs-CRP
levels. Overweight individuals with hypertension, dyslipidemia, and
diabetes had a high prevalence of elevated hs-CRP levels in both sexes.
Japanese adults have very low hs-CRP levels. An aggregation of

metabolic risk factors is associated with elevated hs-CRP levels among
overweight individuals, particularly in women. (C) 2006 Elsevier Ireland Ltd. All rights reserved.

3/7/65 (Item 13 from file: 34)
DIALOG(R)File 34:SciSearch(R) Cited Ref Sci

17064228 Genuine Article#: 225TC Number of References: 81
Title: Genetic factors in diabetic nephropathy
Author(s): Freedman BI (REPRINT) ; Bostrom M; Daeihagh P; Bowden DW
Corporate Source: Wake Forest Univ,Sch Med, Nephrol Sect, Dept Internal
Med,Med Ctr Blvd/Winston Salem//NC/27157 (REPRINT); Wake Forest Univ,Sch Med, Nephrol Sect, Dept Internal Med,Winston Salem//NC/27157;
Wake Forest Univ,Sch Med, Dept Biochem,Winston Salem//NC/27109; Wake
Forest Univ,Sch Med, Ctr Human Genom,Winston Salem//NC/27109
Journal: CLINICAL JOURNAL OF THE AMERICAN SOCIETY OF NEPHROLOGY, 2007, V2,
N6 (NOV), P1306-1316
ISSN: 1555-905X Publication date: 20071100
Publisher: AMER SOC NEPHROLOGY, 1725 I ST, NW STE 510, WASHINGTON, DC 20006
USA
Language: English Document Type: REVIEW
Abstract: Several genes that predispose to type 2 diabetes have recently
been identified. In addition to the recognized and powerful effects of
environmental factors, there is abundant evidence in support of genetic
susceptibility to the microvascular complication of nephropathy in individuals with both type 1 and type 2 diabetes. Familial aggregation of phenotypes such as end-stage renal disease, albuminuria, and chronic kidney disease have routinely been reported in
populations throughout the world, and heritability estimates for albuminuria and glomerular filtration rate demonstrate strong contributions of inherited factors. Recent genome-wide linkage scans
have identified several chromosomal regions that likely contain diabetic nephropathy susceptibility genes, and association analyses
have evaluated positional candidate genes under these linkage peaks.
These complimentary approaches have demonstrated that polymorphisms in
the carnosinase 1 gene on chromosome 18q, the adiponectin gene on

3q, and the engulfment and cell motility gene on 7p are likely associated with susceptibility to diabetic nephropathy. Additional genes that seem to be of importance in renal phenotypes include manganese superoxide dismutase and angiotensin 1-converting enzyme, with nitric oxide synthase implicated in albuminuria. This article reviews the inherited aspects of diabetic kidney disease with particular emphasis on recently implicated genes and pathways. It seems likely that the risk for diabetes-associated kidney disease is magnified by inheriting risk alleles at several susceptibility loci, in the presence of hyperglycemia.

3/7/66 (Item 14 from file: 34)
DIALOG(R)File 34:SciSearch(R) Cited Ref Sci

16967537 Genuine Article#: 213PM Number of References: 144
Title: Strategies to reduce vascular risk associated with obesity
Author(s): Bodary PF (REPRINT) ; Lglay HB; Eitzman DT
Corporate Source: 410 W Warren Ave,3009 Sci Hall/Detroit//MI/48202 (REPRINT); Wayne State Univ,Coll Liberal Arts & Sci, Dept Nutr & Food Sci,Detroit//MI/48202; Univ Michigan,Dept Internal Med, Div Cardiovasc Med,Ann Arbor//MI/48109
Journal: CURRENT VASCULAR PHARMACOLOGY, 2007, V5, N4 (OCT), P249-258
ISSN: 1570-1611 Publication date: 20071000
Publisher: BENTHAM SCIENCE PUBL LTD, EXECUTIVE STE Y26, PO BOX 7917, SAIF ZONE, 1200 BR SHARJAH, U ARAB EMIRATES
Language: English Document Type: REVIEW
Abstract: The obesity pandemic will likely have a significant impact on the global incidence of cardiovascular disease. Although the mechanisms linking obesity and cardiovascular disease are unclear, recent studies have implicated the adipocyte as a potentially important mediator of vascular complications. The adipocyte is no longer considered a passive storage depot for triglycerides and fatty acids, but rather an active metabolic organ capable of producing several factors, commonly referred to as adipokines, that may have effects on many physiological and pathophysiological processes. With increasing fat mass, several adipose-related factors are upregulated that may affect local and distant inflammatory processes, including atherothrombosis. Other

factors, such as adiponectin, are downregulated with increasing fat mass. Although most adipokines are thought to promote vascular disease, several studies over the past few years indicate adiponectin is actually protective against both diabetes and vascular disease. There are now available pharmacologic agents capable of altering the adipocyte transcription profile. This review will focus on the potential impact of adipocyte-derived factors towards vascular disease and emerging therapeutic strategies that may alter these effects.

3/7/67 (Item 15 from file: 34)
DIALOG(R)File 34:SciSearch(R) Cited Ref Sci

16808853 Genuine Article#: 206KE Number of References: 21
Title: Low molecular weight adiponectin negatively correlates with the waist circumference and monocytic IL-6 release
Author(s): Schober F; Neumeler M; Weigert J; Wurm S; Wanninger J; Schaffler A; Dada A; Liebisch G; Schmitz G; Aslanidis C; Buechler C (REPRINT)
Corporate Source: Regensburg Univ Hosp,Dept Internal Med 1,D-93042 Regensburg//Germany/ (REPRINT); Regensburg Univ Hosp,Dept Internal Med 1,D-93042 Regensburg//Germany/; Regensburg Univ Hosp,Inst Clin Chem,D-93042 Regensburg//Germany/; Regensburg Univ Hosp,Lab Med,D-93042 Regensburg//Germany/
Journal: BIOCHEMICAL AND BIOPHYSICAL RESEARCH COMMUNICATIONS, 2007, V361, N4 (OCT 5), P968-973
ISSN: 0006-291X Publication date: 20071005
Publisher: ACADEMIC PRESS INC ELSEVIER SCIENCE, 525 B ST, STE 1900, SAN DIEGO, CA 92101-4495 USA
Language: English Document Type: ARTICLE
Abstract: Adiponectin circulates as trimer (LMW), hexamer (MMW) and high molecular weight multimer (HMW) but the distribution and effects of these isoforms have not been studied in detail. Monocytes were isolated from normal weight and overweight controls and patients with type 2 diabetes mellitus (T2D) and monocytic release of IL-6 positively correlated with the body mass index (BMI). HMW-adiponectin further enhanced and LMW-adiponectin reduced IL-6 release in monocytes. Systemic total adiponectin, and the HMW isoform were not different in these groups but MMW-adiponectin was lower in T2D, and LMW-adiponectin was

reduced in the obese and T2D. Circulating LMW-adiponectin negatively correlated to monocytic IL-6 release. Systemic IL-6 was higher in the obese control group and T2D, respectively, but did not correlate with monocytic IL-6 secretion. Therefore, the current study indicates that HMW-adiponectin exerts pro- and LMW-adiponectin antiinflammatory effects and reduced LMW-adiponectin in obesity may partly contribute to elevated monocytic IL-6 release. (C) 2007 Elsevier Inc. All rights reserved.

3/7/68 (Item 16 from file: 34)
DIALOG(R)File 34:SciSearch(R) Cited Ref Sci

16658303 Genuine Article#: 183XL Number of References: 123
Title: Inflammatory responses underlying the microvascular dysfunction associated with obesity and insulin resistance
Author(s): Singer G; Granger DN (REPRINT)
Corporate Source: Louisiana State Univ,Hlth Sci Ctr, Dept Mol & Cellular Physiol,1501 Kings Highway/Shreveport//LA/71130 (REPRINT); Louisiana State Univ,Hlth Sci Ctr, Dept Mol & Cellular Physiol,Shreveport//LA/71130; Med Univ Graz,Dept Pediat Surg,Graz//Austria/
Journal: MICROCIRCULATION, 2007, V14, N4-5, P375-387
ISSN: 1073-9688 Publication date: 20070000
Publisher: TAYLOR & FRANCIS INC, 325 CHESTNUT ST, SUITE 800, PHILADELPHIA, PA 19106 USA
Language: English Document Type: REVIEW
Abstract: Obesity is a growing health care problem that is increasing the incidence and morbidity of cardiovascular diseases. Emerging evidence suggests that obesity is associated with a systemic inflammatory response that is characterized by endothelial cell dysfunction, oxidative stress, and the activation of circulating immune cells. Adipocytes produce and release a variety of cytokines (IL-1, TNF-alpha) and cytokine-like substances (leptin, resistin) that appear to mediate the inflammatory response that accompanies obesity. The abrogating influence of weight loss on the inflammatory response supports this contention. The insulin resistance that often accompanies obesity may also contribute to this inflammatory phenotype. Studies in experimental

animals and clinical studies suggest that the microvascular dysfunction
associated with pathological states, such as sepsis, is greatly exacerbated by obesity. Although the microvasculature appears to be a
major target for the deleterious inflammatory consequences of obesity,
relatively little attention has been devoted to characterizing the effects of obesity on inflammatory responses in different regional vascular beds and to defining the mechanisms that underlie the resultant microvascular dysfunction.

3/7/69 (Item 17 from file: 34)
DIALOG(R)File 34:SciSearch(R) Cited Ref Sci

16559232 Genuine Article#: 172TU Number of References: 97
Title: Adipokines and coronary vasomotor dysfunction
Author(s): Knudson JD; Dick GM; Tune JD (REPRINT)
Corporate Source: Indiana Univ,Sch Med, Dept Cellular & Integrat Physiol,635 Barnhill Dr/Indianapolis//IN/46202 (REPRINT); Indiana Univ,Sch Med, Dept Cellular & Integrat
Physiol,Indianapolis//IN/46202;
Louisiana State Univ,Hlth Sci Ctr, Dept Physiol,New Orleans//LA/70112
Journal: EXPERIMENTAL BIOLOGY AND MEDICINE, 2007, V232, N6 (JUN), P727-736
ISSN: 1535-3702 Publication date: 20070600
Publisher: SOC EXPERIMENTAL BIOLOGY MEDICINE, 195 WEST SPRING VALLEY AVE,
MAYWOOD, NJ 07607-1727 USA
Language: English Document Type: REVIEW
Abstract: Research in the last 10-15 years has shown that fat cells (adipocytes) produce and release proteins with specific biologic activities. These proteins, termed adipokines, include the hormones
leptin, adiponectin, and resistin. Adipose tissue is now recognized as an active endocrine organ. With the obesity pandemic swelling in the Western world, ongoing research is aimed at determining
the biologic links between obesity and cardiovascular disease. This
review presents basic historical background information on the major
adipokines, introduces findings from clinical studies associating adipokines with cardiovascular disease, and summarizes results from
recent basic science research studies of mechanisms of adipokine-induced cardiovascular dysfunction. Particular emphasis is
placed on the action of adipokines in the coronary

circulation-especially effects of adipokines on endothelial function,
as endothelial damage is likely a critical event initiating atherosclerotic coronary artery disease.

3/7/70 (Item 18 from file: 34)
DIALOG(R)File 34:SciSearch(R) Cited Ref Sci

15150084 Genuine Article#: 041GJ Number of References: 48
Title: The role of adiponectin in atherosclerosis and thrombosis
Author(s): Ekmekci H (REPRINT) ; Ekmekci OB
Corporate Source: Miralay Hasan Kazim Sok,Ersevenler Sit,Mehtap Apt/TR-34290 Istanbul//Turkey/ (REPRINT); Univ Istanbul,Fac Med, Dept Pediat Haematol & Oncol,Istanbul//Turkey/; Univ Istanbul,Bone Marrow Transplantat Unit, Cerrahpasa Med Fac, Dept Biochem,Istanbul//Turkey/(
hakekmekci@yahoo.com)
Journal: CLINICAL AND APPLIED THROMBOSIS-HEMOSTASIS, 2006, V12, N2 (APR), P 163-168
ISSN: 1076-0296 Publication date: 20060400
Publisher: WESTMINSTER PUBL INC, 708 GLEN COVE AVE, GLEN HEAD, NY 11545 USA
Language: English Document Type: ARTICLE
Abstract: Obesity is a major risk factor for morbidity and mortality from cardiovascular causes. Adiponectin has been identified recently as one of the adipocytokines with important metabolic effects. it can suppress atherogenesis by inhibiting the adherence of monocytes, reducing their phagocytic activity, and suppressing the accumulation of modified lipoproteins in the vascular wall. In addition, as adiponectin decrease endothelial damage and stimulates production of NO from vascular endothelial cells, hypoadiponectinemia may be partially contribute to thrombus formation.

3/7/71 (Item 19 from file: 34)
DIALOG(R)File 34:SciSearch(R) Cited Ref Sci

14437979 Genuine Article#: 971NH Number of References: 118
Title: Treating insulin resistance in type 2 diabetes with metformin and thiazolidinediones
Author(s): Bailey CJ (REPRINT)
Corporate Source: Aston Univ,Sch Life & Hlth Sci,Birmingham B4 7ET/W

Midlands/England/ (REPRINT); Aston Univ,Sch Life & Hlth Sci,Birmingham
B4 7ET/W Midlands/England/(c.j.bailey@aston.ac.uk)
Journal: DIABETES OBESITY & METABOLISM, 2005, V7, N6 (NOV), P675-691
ISSN: 1462-8902 Publication date: 20051100
Publisher: BLACKWELL PUBLISHING, 9600 GARSINGTON RD, OXFORD OX4 2DQ, OXON,
ENGLAND
Language: English Document Type: REVIEW
Abstract: Insulin resistance underlies the pathogenesis of hyperglycaemia
and cardiovascular disease in most people with type 2 diabetes.
Metformin and thiazolidinediones (pioglitazone and rosiglitazone)
counter insulin resistance by different cellular mechanisms and with
complementary effects, making them suited for use in combination.
Metformin exerts a stronger suppression of hepatic glucose output,
while thiazolidinediones produce a greater increase in peripheral
glucose uptake, enabling metformin-thiazolidinedione combinations to
improve glycaemic control in type 2 diabetes with additive efficacy.
Basal insulin concentrations are not raised by metformin or
thiazolidinediones, so there is minimal risk of hypoglycaemia, and
metformin can reduce the weight gain associated with
thiazolidinediones. There are overlapping effects of metformin and
thiazolidinediones against a range of athero-thrombotic factors and
markers. These include decreased plasminogen activator inhibitor-1,
reduced platelet aggregation, reductions of several vascular
adhesion molecules, and reduced markers of low-grade inflammation such
as C-reactive protein. Additionally, thiazolidinediones increase
adiponectin and slightly reduce blood pressure. Both metformin
and thiazolidinediones can improve components of the lipid profile:
thiazolidinediones consistently reduce free fatty acid concentrations
and decrease the proportion of small dense low-density-lipoprotein, and
pioglitazone also decreases triglycerides. During co-administration,
metformin and thiazolidinediones do not interfere with each other's
pharmacokinetics, and lower doses of the two agents together can
achieve efficacy with fewer side effects. Metformin-thiazolidinedione
combinations require attention to the precautions for both agents,
especially renal, cardiac and hepatic status. Thus, metformin and
thiazolidinediones can be used in combination to address the
hyperglycaemia and vascular risk in type 2 diabetes. .

3/7/72 (Item 20 from file: 34)
DIALOG(R)File 34:SciSearch(R) Cited Ref Sci

14332480 Genuine Article#: 961LZ Number of References: 38
Title: Mouse and human resistins impair glucose transport in primary mouse
cardiomyocytes, and oligomerization is required for this biological
action
Author(s): Graveleau C; Zaha VG; Mohajer A; Banerjee RR; Dudley-Rucker N;
Steppan CM; Rajala MW; Scherer PE; Ahima RS; Lazar MA; Abel ED (REPRINT)
Corporate Source: Univ Utah,Sch Med, Div Endocrinol Diabet & Metab,15 North
2030 East,Bldg 533,Rm 3410B/Salt Lake City//UT/84112 (REPRINT); Univ
Utah,Sch Med, Div Endocrinol Diabet & Metab,Salt Lake City//UT/84112;
Univ Utah,Program Human Mol Biol & Genet,Salt Lake City//UT/84112; Univ
Penn,Sch Med, Div Endocrinol Diabet & Metab,Philadelphia//PA/19104;
Yeshiva Univ Albert Einstein Coll Med,Dept Cell Biol,Bronx//NY/10461(
dale.abel@hmbg.utah.edu)
Journal: JOURNAL OF BIOLOGICAL CHEMISTRY, 2005, V280, N36 (SEP 9), P 31679-31685
ISSN: 0021-9258 Publication date: 20050909
Publisher: AMER SOC BIOCHEMISTRY MOLECULAR BIOLOGY INC, 9650 ROCKVILLE PIKE, BETHESDA, MD 20814-3996 USA
Language: English Document Type: ARTICLE
Abstract: The adipocytokine resistin impairs glucose tolerance and insulin
sensitivity in rodents. Here, we examined the effect of resistin on
glucose uptake in isolated adult mouse cardiomyocytes. Murine resistin
reduced insulin-stimulated glucose uptake, establishing the heart as a
resistin target tissue. Notably, human resistin also impaired insulin
action in mouse cardiomyocytes, providing the first evidence that human
and mouse resistin homologs have similar functions. Resistin is a
cysteine-rich molecule that circulates as a multimer of a dimeric
form dependent upon a single intermolecular disulfide bond, which, in
the mouse, involves Cys(26); mutation of this residue to alanine (C26A)

produces a monomeric molecule that appears to be bioactive in the liver. Remarkably, unlike native resistin, monomeric C26A resistin had no effect on basal or insulin-stimulated glucose uptake in mouse cardiomyocytes. Resistin impairs glucose uptake in cardiomyocytes by mechanisms that involve altered vesicle trafficking. Thus, in cardiomyocytes, both mouse and human resistins directly impair glucose transport; and in contrast to effects on the liver, these actions of resistin require oligomerization.

3/7/73 (Item 1 from file: 45)
DIALOG(R)File 45:EMCare

0005540660 EMCARE No: 354470016
Exendin-4 improves glycemic control, ameliorates brain and pancreatic pathologies, and extends survival in a mouse model of huntington's disease

Martin B.; Golden E.; Carlson O.D.; Pistell P.; Zhou J.; Kim W.; Frank B.P.; Thomas S.; Chadwick W.A.; Greig N.H.; Bates G.P.; Sathasivam K.; Bernier M.; Maudsley S.; Mattson M.P.; Egan J.M.
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Diabetes (Diabetes) (United States) February 1, 2009, 58/2 (318-328)
PUBLISHER: American Diabetes Association Inc.
CODEN: DIAEA ISSN: 0012-1797 eISSN: 1939-327X
DOI: 10.2337/db08-0799
URL: http://diabetes.diabetesjournals.org/cgi/reprint/58/2/318
DOCUMENT TYPE: Journal; Article RECORD TYPE: Abstract
LANGUAGE: English SUMMARY LANGUAGE: English
NUMBER OF REFERENCES: 50

OBJECTIVE-The aim of this study was to find an effective treatment for the genetic form of diabetes that is present in some Huntington's disease patients and in Huntington's disease mouse models. Huntington's disease is a neurodegenerative disorder caused by a polyglutamine expansion within the

huntingtin protein. Huntington's disease patients exhibit neuronal dysfunction/degeneration, chorea, and progressive weight loss. Additionally, they suffer from abnormalities in energy metabolism affecting
both the brain and periphery. Similarly to Huntington's disease patients,
mice expressing the mutated human huntingtin protein also exhibit neurodegenerative changes, motor dysfunction, perturbed energy metabolism,
and elevated blood glucose levels. RESEARCH DESIGN AND METHODS-Huntington's
disease mice were treated with an FDA-approved antidiabetic glucagon-like
peptide 1 receptor agonist, exendin-4 (Ex-4), to test whether euglycemia
could be achieved, whether pancreatic dysfunction could be alleviated, and
whether the mice showed any neurological benefit. Blood glucose and insulin
levels and various appetite hormone concentrations were measured during the
study. Additionally, motor performance and life span were quantified and
mutant huntingtin (mhtt) aggregates were measured in both the pancreas and brain. RESULTS-Ex-4 treatment ameliorated abnormalities in
peripheral glucose regulation and suppressed cellular pathology in both
brain and pancreas in a mouse model of Huntington's disease. The treatment
also improved motor function and extended the survival time of the Huntington's disease mice. These clinical improvements were correlated with
reduced accumulation of mhtt protein aggregates in both islet and brain cells. CONCLUSIONS-Targeting both peripheral and neuronal deficits,
Ex-4 is an attractive agent for therapeutic intervention in Huntington's
disease patients suffering from diabetes. (c) 2009 by the American Diabetes
Association.

3/7/74 (Item 2 from file: 45)
DIALOG(R)File 45:EMCare

0005474035 EMCARE No: 352753857
Endocrine Functions of Adipose Tissue: Focus on Adiponectin
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University of Missouri-Columbia School of Medicine, Departments of Medicine, Pharmacology, and Physiology Diabetes, Cardiovascular Center of

Excellence Harry S. Truman VA Medical Center, Columbia, MO, United States
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CORRESP. AUTHOR/AFFIL: Sowers J.R.: University of Missouri-Columbia School of Medicine, Departments of Medicine, Pharmacology, and Physiology
Diabetes, Cardiovascular Center of Excellence Harry S. Truman VA Medical
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2008, 9/1 (32-40)
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DOI: 10.1016/S1098-3597(08)60026-5
DOCUMENT TYPE: Journal; Article RECORD TYPE: Abstract
LANGUAGE: English SUMMARY LANGUAGE: English
NUMBER OF REFERENCES: 50

Accumulating evidence indicates that obesity and overweight are associated with, and contribute to, the development of type 2 diabetes mellitus (DM), cardiovascular disease (CVD), and chronic kidney disease
(CKD). The adipocyte-derived cytokine, adiponectin, has been shown to improve insulin sensitivity, increase rates of fatty acid oxidation, decrease muscle lipid content, and reduce inflammation and vascular injury.
However, adiponectin levels have been found to be reduced in persons with obesity and type 2 DM. Furthermore, adiponectin levels are inversely associated with those of tumor necrosis factor-alpha and C-reactive protein-markers of endothelial dysfunction and systemic inflammation. The 2 receptors for adiponectin-Adipo R SUB 1 and Adipo R SUB 2 , which are expressed in muscle and liver tissue and in human fat
cells-are hormonally regulated, with increased insulin levels causing a
reduction in their abundance. The hyperinsulinemia observed in obesity,
therefore, may be partially responsible for the reduction in the numbers of
adiponectin receptors. Adiponectin aggregates range from
a hexamer of low molecular weight to larger multimeric structures of high
molecular weight. A smaller proteolytic fragment-the globular head domain
of adiponectin, or gAd-interacts specifically with skeletal muscle. The relation of circulating adiponectin to its biologic actions is more complex than originally believed; therefore, it is the multimeric forms of the adiponectin molecule that need to be measured and

evaluated in relation to associated metabolic, cardiovascular, and renal
functions. Furthermore, strategies to measure the numbers of adiponectin receptors on available tissue need to be developed to fully assess the clinical role of adiponectin in type 2 DM, CVD, and CKD. (c) 2008 Excerpta Medica.

3/7/75 (Item 3 from file: 45)
DIALOG(R)File 45:EMCare

0005326099 EMCARE No: 351486145
Alcohol consumption and heart failure: A systematic review
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Current Atherosclerosis Reports (Curr. Atheroscler. Rep.) (United Kingdom) April 1, 2008, 10/2 (117-120)
PUBLISHER: Current Science Ltd
CODEN: CARUC ISSN: 1523-3804
DOI: 10.1007/s11883-008-0017-z
DOCUMENT TYPE: Journal; Review RECORD TYPE: Abstract
LANGUAGE: English SUMMARY LANGUAGE: English
NUMBER OF REFERENCES: 45

Heart failure (HF) remains a major public health issue. It is estimated
that about 500,000 Americans per year are diagnosed with HF. Despite advanced medical and surgical treatments for HF, mortality after the onset
of HF is still high, thereby underscoring the importance of primary prevention. Among modifiable lifestyle factors, alcohol consumption appears
to play a role in the development of HF. Although excessive drinking has
been known to lead to alcoholic cardiomyopathy and light-to-moderate drinking may confer some cardiovascular benefits, recent studies suggest it
is not only the quantity, but also drinking patterns and genetic factors,
that may influence the relation between alcohol consumption and cardiovascular disease. This article reviews current evidence on the

association between alcohol consumption and HF. Copyright (c) 2008 by Current Medicine Group LLC.

3/7/76 (Item 4 from file: 45)
DIALOG(R)File 45:EMCare

0004449621 EMCARE No: 38680638
Metabolic syndrome: An appraisal of the pro-inflammatory and procoagulant status
Devaraj S.; Rosenson R.S.; Jialal I.
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Endocrinology and Metabolism Clinics of North America (Endocrinol. Metabolism Clin. North Am.) (United States) June 1, 2004, 33/2 (431-453)
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Inflammation and hypercoagulability predispose to atherothrombosis and seem to be important features of the metabolic syndrome. The most convincing evidence is the association with increased levels of C-reactive protein. The hemostatic abnormality that has been most consistently associated with insulin resistance is an elevated plasminogen activator inhibitor-1 level. In contrast, markers of hypercoagulability have been associated inconsistently with hyperinsulinemia and glucose intolerance. Fibrinogen clusters with inflammatory factors, which suggests involvement of adipose tissue-generated inflammatory cytokines. Elevated von Willebrand's factor and factor VIII levels aggregate with indicators of endothelial injury, whereas vitamin K-dependent coagulation proteins

correlate with triglyceride levels.

3/7/77 (Item 1 from file: 65)
DIALOG(R)File 65:Inside Conferences

05134031 INSIDE CONFERENCE ITEM ID: CN053441735
Enhanced Platelet Aggregation and Thrombogenic Tendency in Adiponectin-Deficient Mice. (8:15 AM)
Kato, H.; Kashiwagi, H.; Shiraga, M.; Honda, S.; Miyata, S.; Yamamoto, J.; Kurata, Y.; Funahashi, T.; Shimomura, I.; Tomiyama, Y.
CONFERENCE: American Society of Hematology; Abstracts for the 46th annual meeting of the American Society of Hematology-Annual meeting; 46th
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American Society of Hematology, 2004
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3/7/78 (Item 1 from file: 71)
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Anti-inflammatory and cardioprotective effects of n-3 polyunsaturated fatty acids and plant sterols in hyperlipidemic individuals
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DOCUMENT TYPE: Article
LANGUAGES: English SUMMARY LANGUAGES: English
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Background: Risk factors of cardiovascular disease such as lipid aberrations, hypertension, abdominal adiposity and elevations in systemic inflammation, are prominent aetiologies in hyperlipidemia. Supplementation with n-3 PUFA is associated with a reduction in cardiovascular events through its hypotriglyceridemic, anti-aggregatory and anti-inflammatory properties. Plant sterols have potent hypocholesterolemic properties, although their effect on the inflammatory cascade is uncertain. This study investigated the effect of combined supplementation with n-3 PUFA and plant sterols on cardiovascular risk factors, blood pressure, body composition, markers of systemic inflammation and overall risk, in hyperlipidemic individuals. Methods: The study was a 3-week randomised, double-blind, placebo-controlled, 2 x 2 factorial design, in four parallel groups. Sixty hyperlipidemic participants were randomised to recieve either sunola oil or 1.4 g/d n-3 PUFA capsules with or without 2 g plant sterols per day. Results: The combination of n-3 PUFA and plant sterols reduced several inflammatory markers. High sensitivity C-reactive protein (hs-CRP) was reduced by 39% (P = 0.009), tumor necrosis factor-alpha (TNF-alpha) by 10% (P = 0.02), interleukin-6 (IL-6) by 10.7% (P = 0.009), leukotriene B SUB 4 (LTB SUB 4) by 29.5% (P = 0.01) and adiponectin was increased by 29.5% (P = 0.05). Overall cardiovascular risk was reduced by 22.6% (P = 0.006) in the combination group. Conclusion: We have demonstrated, for the first time that dietary intervention with n-3 PUFA and plant sterols reduces systemic inflammation in hyperlipidemic individuals. Furthermore, our results suggest that reducing inflammation provides a potential mechanism by which the combination of n-3 PUFA and plant sterols are cardioprotective.

3/7/79 (Item 1 from file: 72)
DIALOG(R)File 72:EMBASE

0082908217 EMBASE No: 2009145345
Adiponectin and leptin in ischemic stroke
Adiponektyna i leptyna a udar niedokrwienny mozgu

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LANGUAGE: Polish SUMMARY LANGUAGE: Polish; English
NUMBER OF REFERENCES: 54

Abdominal obesity becomes very significant health's problem, especially
because it is connected with pathogenesis of cardiovascular diseases.
Adipose tissue is not only a store of excess energy but a hormonally active
system too. The substances produced by adipose tissue are adipocytokines.
Two of them are leptin and adiponectin. Adiponectin levels are inversely related to the adiposity degree, despite of adipose tissue is
only source of it. Concentrations of adiponectin have been reported
to be decreased in patients with coronary artery diseases, type II diabetes
mellitus, hypertensions and dyslipidemia patients in some insulin resistant
states. It takes part in processes regulate glucose and lipid metabolism
and it has anti-inflammatory and antiatherogenic properties.
Adiponectin has a potential protective ability towards to
cardiovascular diseases. Positive correlation with degree of adiposity has
been reported for leptin - hormone involved in the regulation of food
intake and energy expenditure. Leptin exerts many potentially atherogenic
effects. It has been reported to influence on arterial hypertension,
endothelial dysfunction, platelet aggregation, insulin resistant and
activation of sympathetic system. In this way it can play very important
role in development of stroke. Recent studies suggest that
adiponectin and leptin may play an important role in
obesity-associated cerebrovascular diseases. There is still too little
evidence to say that these two hormones are independent marks of ischemic
stroke and confirm their role in stroke pathogenesis. (c) Aktualn Neurol

2008.

3/7/80 (Item 2 from file: 72)
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0082778227 EMBASE No: 2008598154
Adiponectin-adipocytes-derived hormone and its multidirectional function
Adiponektyna-hormon adipocytarny i jego wieloukladowe znaczenie
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Clinical and Experimental Medical Letters (Clin. Exp. Med. Lett.) (
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LANGUAGE: Polish SUMMARY LANGUAGE: Polish; English
NUMBER OF REFERENCES: 65

Increasing morbidity and mortality from cardiovascular and neoplastic disorders is the effect of more frequent problem of obesity; isulinresistance and type 2 diabetes; not only in developed countries but in developing as well. Excess adipose tissue in particular compartments of human body is connected with numerous risk factors of artherosclerosis such as hyperinsulinaemia, hypertension, dylipidaemia, disregulations of glucose metabolism. Several studies that have been conducted so far, have focused on the patomechanism of mentioned disorders with a particular attention paid to metabolism of adipocytes. Plasma proteins derived from adipocytes have been observed, especially the one called adiponectin. Adiponectin is the protein product of apM1 gene expression. Identified receptors of adiponectin: AdipoR1 and AdipoR2 are localised not only on adipocytes but also on hepatocytes and skeletal myocytes. Activation of the pleiotropic enzyme AMP-dependent protein kinase

is integral to the signalling intracellular effects of adiponectin. The potential antiinflammatory and antiartherosclerotic effects of adiponectin activity, as well as inhibition of neoplastic tranformation originate from its influence on cellular adhesion molecules
presentation and cytokines and growth factors production. Adiponectin, adipocytes-derived protein, appears in circulation in a form of hexamer
or multimer. Considering the current data, adiponectin is an integral part of several signalling pathways of alive cell. There is obvious necessity of further researches on the precise role of adiponectin and prospective use of gained knowledge in clinical practice.

3/7/81 (Item 3 from file: 72)
DIALOG(R)File 72:EMBASE

0082546606 EMBASE No: 2008378719
Current studies on therapeutic approaches for ischemia/reperfusion injury
in steatotic livers
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38/9 (851-859)
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DOCUMENT TYPE: Journal; Review RECORD TYPE: Abstract
LANGUAGE: English SUMMARY LANGUAGE: English
NUMBER OF REFERENCES: 86

Steatotic livers are particularly vulnerable to ischemia/reperfusion (I/
R) injury, resulting in poor outcomes following liver surgery and transplantation. Therapeutic approaches for I/R injury in steatotic livers
are currently under intensive investigation. This review summarizes and
discusses the approaches developed during the last few years to prevent
hepatic I/R injury in steatotic livers. Among the proposed approaches,

ischemic preconditioning and intermittent clamping are the two most promising approaches that have been applied in some clinical centers for
liver surgery and transplantation, but most of others have not reached clinical application yet. (c) 2008 The Japan Society of Hepatology.

3/7/82 (Item 4 from file: 72)
DIALOG(R)File 72:EMBASE

0082522990 EMBASE No: 2008316157
Rimonabant in rats with a metabolic syndrome: Good news after the depression
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July 1, 2008, 154/5 (915-917)
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PUBLISHER ITEM IDENTIFIER: BJP2008170
DOI: 10.1038/bjp.2008.170
DOCUMENT TYPE: Journal; Note RECORD TYPE: Abstract
LANGUAGE: English SUMMARY LANGUAGE: English
NUMBER OF REFERENCES: 12

The synthetic cannabinoid CB SUB 1 receptor antagonist rimonabant (sold
in the United Kingdom under the brand name Acomplia) was reported to improve the profile of cardiovascular risk factors in obese patients with
the metabolic syndrome, a cluster of metabolic disorders that often precedes the onset of type II diabetes. Rimonabant is shown in the current
issue of British Journal of Pharmacology to attenuate weight gain in Zucker
rats, an experimental model of insulin resistance. Neutrophil and monocyte
counts were lowered by rimonabant administration. Both platelet activation
(by ADP) and aggregation (in response to thrombin) were inhibited. Circulating pro-inflammatory cytokine levels (monocyte chemotactic protein
1, MCP1 and Regulated upon Activation, Normal T-cell Expressed and

Secreted, RANTES) were also reduced. Furthermore, fibrinogen levels returned to normal. These favourable anti-inflammatory and anti-thrombotic
actions imply for rimonabant a peripheral, direct action on some cardiovascular risk factors. (c) 2008 Nature Publishing Group All rights
reserved.

3/7/83 (Item 5 from file: 72)
DIALOG(R)File 72:EMBASE

0082485431 EMBASE No: 2008329961
Genetic epidemiology of diabetic retinopathy
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URL: http://www.expert-reviews.com/doi/pdf/10.1586/17469899.3.2.165
DOCUMENT TYPE: Journal; Review RECORD TYPE: Abstract
LANGUAGE: English SUMMARY LANGUAGE: English
NUMBER OF REFERENCES: 128

Diabetic retinopathy (DR) is the leading cause of vision loss and blindness among adults in developed countries. Despite intensive antidiabetic treatment, the global prevalence of DR is growing due to an
increasing incidence and prolonged survival of diabetic individuals. As a
complex disease, DR is a consequence of multiple interactions between environmental and genetic factors. Racial differences in incidence, familial aggregation and candidate gene association studies suggest that genetic factors play a role in the etiology of DR. Despite approximately 30 years of research, the molecular genetics of DR is still
in its infancy owing to the low quality of most research studies. To date,
only a few genetic markers from one gene, AKR1B1 (alclose reductase) - the

first enzyme involved in the polyol pathway of glucose metabolism that converts glucose into sorbitol - have been identified. Successful genetic
epidemiology requires a high sample size, longitudinal designs, analysis of
large haplotype blocks integrated by tag single nucleotide polymorphisms
identified by means of human haplotype map (HapMap) data, genome-wide association studies using high density microarray-based single nucleoticle
polymorphism genotyping and pharmacogenomic approaches. An urgent move from
old genetics to modern genomics is necessary to boost the ability to identify genes contributing to the development and progression of DR. (c)
2008 Future Drugs Ltd.

3/7/84 (Item 6 from file: 72)
DIALOG(R)File 72:EMBASE

0082269058 EMBASE No: 2008063117
Effects of olmesartan on endothelial function
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DOCUMENT TYPE: Journal; Review RECORD TYPE: Abstract
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NUMBER OF REFERENCES: 72

It is well established that a functional endothelium contributes to maintain cardiovascular homeostasis mainly through the activity of endothelium-derived nitric oxide (NO). However, in the presence of proatherogenic risk factors including hypertension, diabetes mellitus and
hypercholesterolaemia, the bioavailability of NO is reduced. This condition

is defined as endothelial dysfunction and characterised by vasoconstriction, platelet aggregation, leucocyte adhesion and smooth muscle cell proliferation. A reduced availability of NO is mainly due to an increase in reactive oxygen species (ROS) production, which is responsible for NO breakdown. A large body of evidence indicates that, especially under pathological conditions, the activity of the renin-angiotensin system (RAS) is associated with angiotensin II (Ang II)-mediated ROS production, thus unbalancing endothelial function and leading to progressive vascular disease. The action of RAS is mostly linked to the downstream effects of the binding with the Ang II subtype 1 receptors (AT1). Therefore, selective RAS blockade with angiotensin receptor blockers (ARBs) is able to restore endothelial function in patients with cardiovascular risk factors. Olmesartan, an effective ARB, beyond its blood-pressure lowering effect, has been reported to affect the redox state of the vessel wall by restoring NO availability under different pathological conditions. Furthermore, it has been described that olmesartan exerts anti-inflammatory effects and increases endothelial progenitor cells. This article reviews the evidence linking olmesartan to vascular endothelial protection and examines the possibility that this effect translates to beneficial clinical properties of this ARB. (c) 2007 Adis Data Information BV. All rights reserved.

3/7/85 (Item 7 from file: 72)
DIALOG(R)File 72:EMBASE

0081835597 EMBASE No: 2007269702
Primary culture of human omental preadipocytes and study of their biological properties
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National Medical Journal of China (Nat. Med. J. China) (China) March
27, 2007, 87/12 (838-841)
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DOCUMENT TYPE: Journal; Article RECORD TYPE: Abstract
LANGUAGE: Chinese SUMMARY LANGUAGE: Chinese; English
NUMBER OF REFERENCES: 12

Objective: To develop a primary culture method of human omental preadipocytes and to study their biological properties, such as hyperplasia, hypertrophy and endocrine secretion of human visceral adipose tissue. Methods: Using enzyme-digesting method, fibroblast-like cells from the human omental adipose tissues were cultured. The morphological changes of the cultured cells were observed and the growth curve was drawn by MTT method. The intracytoplasmic lipid of the cultured cells was determined by oil red O staining. The leptin and adiponectin levels in the culture supernatants were measured by ELISA. Results: The cultured fibroblast-like cells were homogeneous. Proliferation of cells began at the 3 rd day and the cell numbers increased in indicial way from the 3 rd day to the 9 th day. The doubling time of cells was about 60 hours. During the process of induction by conditional medium, the cells became round and larger, and more adipose droplets were aggregated. On the 21 st day, more than 90% of the cells became adipocytes. Leptin secretion was detected at low level in the preadipocytes and continuously increased during differentiation, with a peak on day 17. It remained constant from day 17 onward. Unlike leptin, adiponectin secretion was not detected until day 7 after induction, when differentiated adipocytes had already been observed. Its secretion increased dramatically between days 7 and 17, and reached a maximum level on day 17, but had a significant reduction on day 21. Extraction of intracytoplasmic lipid stained with oil red O and detection of leptin and adiponectin both verified the isolated cells were preadipocytes functioning actively. Conclusion: A human omental preadipocytes model has been established and different secretion patterns of leptin and adiponectin secretion related to preadipocyte differentiation has been characterized. Adiponectin may be proposed as a specific marker for preadipocyte differentiation.

3/7/86 (Item 8 from file: 72)
DIALOG(R)File 72:EMBASE

0081309140 EMBASE No: 2006371567
Targeting adiponectin for cardioprotection
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United Kingdom) August 1, 2006, 10/4 (573-581)
CODEN: EOTTA ISSN: 1472-8222
DOI: 10.1517/14728222.10.4.573
DOCUMENT TYPE: Journal; Review RECORD TYPE: Abstract
LANGUAGE: English SUMMARY LANGUAGE: English
NUMBER OF REFERENCES: 106

Adiponectin is an adipose tissue-derived plasma protein which has a reduced concentration in subjects with obesity-related diseases. Adiponectin has anti-diabetic and anti-inflammatory characteristics, which lead to beneficial actions on various obesity-linked complications.
Recent experimental findings have shown that adiponectin contributes to protection against cardiac remodelling after pressure overload and cardiac injury following ischaemia-reperfusion. Thus, adiponectin could emerge as a potential cardioprotective agent for the treatment of
several pathological heart conditions. (c) 2006 Informa UK Ltd.

3/7/87 (Item 9 from file: 72)
DIALOG(R)File 72:EMBASE

0081172346 EMBASE No: 2006234601
Adiponectin: Vascular protection from the fat?
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PUBLISHER ITEM IDENTIFIER: 000436052006020000001
DOI: 10.1161/01.ATV.0000200222.55680.df
DOCUMENT TYPE: Journal; Editorial RECORD TYPE: Citation
LANGUAGE: English
NUMBER OF REFERENCES: 19

3/7/88 (Item 10 from file: 72)
DIALOG(R)File 72:EMBASE

0081136820 EMBASE No: 2006198770
Adipocyte: A potential target for the treatment of atherosclerosis
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PUBLISHER ITEM IDENTIFIER: S0306987706000508
DOI: 10.1016/j.mehy.2005.12.042
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LANGUAGE: English SUMMARY LANGUAGE: English
NUMBER OF REFERENCES: 48

Obesity is an independent risk factor for coronary heart disease, whereas the underlying mechanisms have not been fully elucidated. Adipocytes may produce various adipokines with favorable and unfavorable cardiovascular effects. The dysregulated secretion of adipokines by adipocytes may contribute to obese associated atherosclerosis. Adipocytes can also function as phagocytes to uptake and degrade oxidized low-density lipoprotein (Ox-LDL), suggesting that adipocytes possibly involve in

clearance of Ox-LDL in blood. The dysfunctional adipocytes might be implicated in the atherogenesis. Some cardioprotective drugs mediate their
cardiovascular benefits partly through their direct beneficial effects on
adipocytes. Therefore, we hypothesized that adipocytes might be potential
target for the treatment of atherosclerosis. (c) 2006 Elsevier Ltd. All
rights reserved.

0081048235 EMBASE No: 2006108250
Linking inflammation and atherogenesis: Soluble markers identified for
the detection of risk factors and for early risk assessment

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366/1-2 (74-80)
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PUBLISHER ITEM IDENTIFIER: S0009898105006352
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DOCUMENT TYPE: Journal; Review RECORD TYPE: Abstract
LANGUAGE: English SUMMARY LANGUAGE: English
NUMBER OF REFERENCES: 56

Increasing evidence has shown that atherogenesis is not only caused by
hypercholesterolemia. Several risk factors including abdominal obesity,
dyslipidemia, hyperglycemia, bacterial and viral infection, hyperhomocysteinemia have been identified recently, all mediated through

inflammation, which can lead to atherosclerosis. Several events have also
been identified to be involved in the overall inflammation reaction in the
blood vessel which include endothelium dysfunction, expression of adhesion
molecules, recruitment of leukocytes to the injured endothelium, migration
of monocytes to the arterial intima, and transformation of monocytes to
macrophages. In order to facilitate the assessment of early risk for
atherogenesis we have made an effort in this review to identify soluble
markers that will allow the detection of these risk factors and the
identification of associated inflammation events. Since early risks for
atherogenesis are largely preventable with dietary modification and
lifestyle changes, capable of detecting early risks by monitoring soluble
risk markers is conceivably important for asymptomatic individuals to avoid
serious or fatal consequences of atherosclerosis. These soluble markers
should also be useful for monitoring the effectiveness of intervention and
for the identification of therapeutic targets. (c) 2005 Elsevier B.V. All
rights reserved.

3/7/90 (Item 12 from file: 72)
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0081048230 EMBASE No: 2006108245
Genomic variants in polycystic ovary syndrome
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366/1-2 (14-26)
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DOI: 10.1016/j.cca.2005.10.017

DOCUMENT TYPE: Journal; Review RECORD TYPE: Abstract
LANGUAGE: English SUMMARY LANGUAGE: English
NUMBER OF REFERENCES: 181
The polycystic ovary syndrome (PCOS) is a common disorder in premenopausal women, characterized by the presence, among other traits, of hyperandrogenism, insulin resistance, and hyperinsulinism. The familial aggregation of PCOS lead the interest to the molecular genetic basis of this syndrome, especially to the genes encoding proteins involved in androgen synthesis and the regulation of insulin synthesis and action. Considering the relationship between insulin resistance and chronic inflammation, and the clustering of inflammatory markers in PCOS patients, recent studies focused on the involvement of proinflammatory genotypes on the pathogenesis of PCOS. Mounting evidence suggest at present a complex model of inheritance for PCOS, in which predisposing and protecting genomic variants interact with environmental factors such as obesity and a sedentary lifestyle, finally leading to the classic phenotype of this syndrome. Moreover, the association of hyperandrogenism, insulin resistance and chronic inflammation raised the possibility of an increase risk of cardiovascular disease in women suffering from PCOS. In the present review we will summarize the most important findings published to date regarding the molecular genetic mechanisms underlying the association of PCOS with insulin resistance and chronic inflammation, and the possible interaction of these mechanisms with environmental factors.

3/7/91 (Item 13 from file: 72)
DIALOG(R)File 72:EMBASE

0080482469 EMBASE No: 2005126627
Cardiovascular and metabolic disease: New opportunities for therapy
Vlasuk G.P.; Scarborough R.
Dept. of Cardiovasc./Metabolic Dis., Discovery Research, Wyeth Pharmaceuticals, 87 Cambridgepark Drive, Cambridge, MA 02140, United States
CORRESP. AUTHOR/AFFIL: Vlasuk G.P.: Dept. of Cardiovasc./Metabolic Dis., Discovery Research, Wyeth Pharmaceuticals, 87 Cambridgepark Drive,

Cambridge, MA 02140, United States

Current Opinion in Pharmacology (Curr. Opin. Pharmacol.) (United Kingdom) April 1, 2005, 5/2 SPEC. ISS. (119-121)
CODEN: COPUB ISSN: 1471-4892
PUBLISHER ITEM IDENTIFIER: S1471489205000184
DOI: 10.1016/j.coph.2005.02.002
DOCUMENT TYPE: Journal; Editorial RECORD TYPE: Citation
LANGUAGE: English

3/7/92 (Item 14 from file: 72)
DIALOG(R)File 72:EMBASE

0080189180 EMBASE No: 2004368723
Tumor necrosis factor and its potential role in insulin resistance and
nonalcoholic fatty liver disease
Diehl A.M.
The Johns Hopkins Univ. Sch. of Med., Department of Medicine, Div. Gastroenterol., Ross Res. B., Baltimore, MD, United States; Duke University, Genome Science Research Building1, Suite 1073, Box 3256, Res.
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CORRESP. AUTHOR EMAIL: annamae.diehl@duke.edu

Clinics in Liver Disease (Clin. Liver Dis.) (United States) August 1, 2004, 8/3 (619-638)
CODEN: CLDIF ISSN: 1089-3261
PUBLISHER ITEM IDENTIFIER: S108932610400042X
DOI: 10.1016/j.cld.2004.04.012
DOCUMENT TYPE: Journal; Review RECORD TYPE: Abstract
LANGUAGE: English SUMMARY LANGUAGE: English
NUMBER OF REFERENCES: 120

Adipose tissue is an important source of TNF and molecules that regulate TNF activity. Visceral adiposity, in particular, promotes a profile of adipokine expression (ie, low adiponectin, high TNF) that permits high TNF activity and insulin resistance. At the cellular level, TNF-dependent activation of stress-related kinases inhibits insulin signal transduction, causing cellular insulin resistance. Some of the same kinases

also promote further production of TNF, perpetuating a
self-re-enforcing,
positive feedback mechanism for sustained TNF activity and chronic
insulin
resistance. Increased TNF inhibits the hepatic actions of adiponectin
, increasing hepatic fatty acid uptake while reducing hepatic fatty
acid
oxidation and triglyceride export. In aggregate, these responses
cause the net accumulation of fat within hepatocytes (ie, hepatic
steatosis). In mice, the increased delivery of fatty acids to
hepatocytes
is sufficient to induce hepatic insulin resistance. Consistent with
this
finding, it has been shown that TNF-dependent inhibition of
adiponectin blocks normal insulin-mediated suppression of hepatic
glucose output, causing hyperglycemia. This hyperglycemia, in turn,
stimulates hyperinsulinemia. Hyperinsulinemia also attenuates the
further
propagation of insulin-initiated signals (ie, hyperinsulinemic insulin
resistance). Given this evidence that fatty livers participate in the
pathogenesis of type 2 diabetes, therapy to prevent or reverse NAFL
seems
appropriate. Increased TNF also promotes hepatocyte death by inducing
molecules that cause apoptosis and molecules that cause necrosis.
Which
death response occurs in any given hepatocyte may be determined by the
cellular ATP level (low ATP favors necrosis over apoptosis). In
addition,
the increased TNF activity induces the production of other cytokines
and
chemokines that promote the hepatic accumulation of inflammatory
cells.
NASH is the histologic manifestation of increased rates of hepatocyte
death
and associated inflammatory cell infiltration. Studies in experimental
animals prove that TNF-related insulin resistance causes NASH and
alcohol-induced steatohepatitis (ASH), because treatment with agents
that
directly inhibit TNF activity improve insulin resistance, NASH, and
ASH.
Similarly, treatment with insulin sensitizers reduces TNF activity and
improves NASH and ASH. Recent evidence raises the ominous possibility
that
NASH may be a premalignant condition. Chronic exposure to TNF induces
hepatic oxidant stress, and the latter reduces the proliferative
activity
of mature hepatocytes. The increased death rates and reduced
proliferative
activity of mature hepatocytes, in turn, promote liver progenitor
populations to expand. This may set the stage for subsequent hepatic
neoplasia, because in people and experimental animals, the incidence
of

hepatocellular carcinoma is increased in settings that promote the accumulation of hepatic progenitor cells. Nonalcoholic steatohepatitis also
appears to play a permissive role for hepatic fibrosis, because the incidence of cirrhosis is increased in individuals with NASH compared with
those with simple NAFL. Studies in people and experimental animals, however, also clearly demonstrate that NASH is not sufficient to assure the
development of cirrhosis. Indeed many (and perhaps most) individuals with
NASH never develop cirrhosis. Efforts to explain interindividual variability in hepatic fibrosis have identified factors that act on hepatic
stellate cells to regulate hepatic fibrogenesis. These include leptin, certain neurotransmitters (eg, norepinephrine, angiotensin, and acetylcholine) and other cytokines (eg, IL-10 and TGF-beta). Interactions
between TNF and each of these factors have been demonstrated, as have interactions among the factors themselves. Additional research is needed to
clarify the exact milieu required to drive a sustained fibrogenic response
during liver injury. This knowledge has important clinical implications,
because it will help identify the subset of NASH patients who are destined
to become cirrhotic and therefore, most worthy of aggressive clinical intervention.

0080056461 EMBASE No: 2004241636
The chronic inflammatory hypothesis for the morbidity associated with
morbid obesity: Implications and effect of weight loss
Cottam D.R.; Mattar S.G.; Barinas-Mitchell E.; Eid G.; Kuller L.; Kelley
D.E.; Schauer P.R.
Obesity and Nutr. Research Center, Univ. of Pittsburgh Medical Center,
Pittsburgh, PA, United States; Minimally Invasive Surgery Center,
Department of Surgery, Univ. of Pittsburgh Medical Center, Pittsburgh, PA
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Street, Pittsburgh, PA 15213, United States

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Obesity Surgery (Obes. Surg.) (United States) May 1, 2004, 14/5 (589-600)
CODEN: OBSUE ISSN: 0960-8923
DOI: 10.1381/096089204323093345
DOCUMENT TYPE: Journal; Review RECORD TYPE: Abstract
LANGUAGE: English SUMMARY LANGUAGE: English
NUMBER OF REFERENCES: 185

Background: Obesity is a worldwide pandemic that causes a multitude of co-morbid conditions. However, there has been slow progress in understanding the basic pathophysiology that underlies co-morbid conditions associated with obesity. Recently, there has been intense interest in the role of inflammation in obesity. Using the inflammatory hypothesis, many of the mechanisms by which co-morbid conditions are associated with obesity are being elucidated. Methods: We searched the literature and reviewed all relevant articles. We focused on hormones and cytokines that have been associated with other inflammatory conditions such as sepsis and systemic inflammatory response syndrome. Findings: Angiotensinogen (AGT), transforming growth factor beta (TGFbeta), tumor necrosis factor alpha (TMFalpha), and interleukin six (IL-6) are all elevated in obesity and correlate with several markers of adipocyte mass. These mediators have detrimental effects on hypertension, diabetes, dyslipidemia, thromboembolic phenomena, infections, and cancer. Weight loss results in a reduction of inflammatory mediators and a diminution of the associated co-morbid conditions. Conclusions: The success of weight loss surgery in treating the complications associated with obesity is most probably related to the reduction of inflammatory mediators. While some aspects of bariatric physiology remain unclear, there appears to be a strong association between obesity and inflammation, thereby rendering obesity a chronic inflammatory state. A clearer understanding of the physiology of obesity will allow physicians who treat the obese to develop better strategies to promote weight loss and improve the well-being of millions of individuals.

0079995567 EMBASE No: 2004180717
Techniques: Cardiovascular pharmacology and drug discovery in the 21st
century
Douglas S.A.; Ohlstein E.H.; Johns D.G.
Vascular Thrombosis and Inflammation, Cardiovasc. Urogenital Ctr. E.,
GlaxoSmithKline, King of Prussia, PA 19406-0939, United States
AUTHOR EMAIL: steve; a; douglas@gsk.com
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CORRESP. AUTHOR EMAIL: steve; a; douglas@gsk.com

Trends in Pharmacological Sciences (Trends Pharmacol. Sci.) (United
Kingdom) April 1, 2004, 25/4 (225-233)
CODEN: TPHSD ISSN: 0165-6147
PUBLISHER ITEM IDENTIFIER: S0165614704000641
DOI: 10.1016/j.tips.2004.02.013
DOCUMENT TYPE: Journal; Review RECORD TYPE: Abstract
LANGUAGE: English SUMMARY LANGUAGE: English
NUMBER OF REFERENCES: 88
The latter half of the 20th century has been characterized by pharmacologists as the 'age of the receptor', an era in which the bioassay, that stalwart of classical pharmacology, has played a seminal role in identifying novel cardiovascular medicines. In this article, we ask what, if anything, has changed in the pharmacologist's approach to discovering novel cardiovascular drugs on this, the 25th anniversary of the inaugural publication of Trends in Pharmacological Sciences.

3/7/95 (Item 1 from file: 144)
DIALOG(R)File 144:Pascal

19079071 PASCAL No.: 09-0160178
Comparison of Immunoassays for the Selective Measurement of Human High-Molecular Weight Adiponectin
DAN LIU; SCHUSTER Tibor; BAUMANN Marcus; ROOS Marcel; SOLLINGER Daniel;
LUTZ Jens; HEEMANN Uwe; VON EYNATTEN Maximilian
Department of Nephrology, Technische Universitaet Muenchen, Munich, Germany; Department of Endocrinology, the Second Hospital of Sun Yat-Sen
University, Guangzhou, China; Institute of Medical Statistics and Epidemiology, Technische Universitaet Muenchen Ismaningerstr, Munich, Germany

Journal: Clinical chemistry : (Baltimore, Md.), 2009, 55 (3) 568-572
ISSN: 0009-9147 CODEN: CLCHAU Availability: INIST-7603; 354000187401760290
No. of Refs.: 17 ref.
Document Type: P (Serial) ; A (Analytic)
Country of Publication: United States
Language: English
BACKGROUND: Adiponectin is an adipocyte-derived hormone circulating in different multimer complexes. The high-molecular-weight (HMW) complex is likely the active form of this protein and has been recognized as a risk marker for type 2 diabetes and coronary artery disease (CAD). Because quantification of HMW adiponectin by Western blot analysis is time-consuming, novel ELISAs have been developed to simplify measurements in clinical research. However, these enzyme immunoassays have not been cross-validated in larger patient groups. We evaluated 2 individual ELISA systems by comparison to Western blotting for measurement of the distribution of HMW adiponectin in healthy individuals and patients with CAD and type 2 diabetes. METHODS: We measured HMW adiponectin in 204 individuals (83 CAD patients, 81 type 2 diabetes patients, and 40 healthy controls). Correlations, range of agreement, and imprecision of HMW concentrations obtained using 2 commercial ELISAs (#1, ALPCO Diagnostics; #2, Millipore) were evaluated by comparison with quantitative Western blotting. RESULT: Adiponectin results of the ELISAs were significantly correlated with those obtained by Western blotting (both r > 0.75, P < 0.001). Deming regression and Bland-Altman analyses indicated high agreement among the 3 immunoassays. The median difference between HMW adiponectin concentrations measured by ELISA and by Western blot was +0.4 mg/L for ELISA #1 and -0.4 mg/L for ELISA #2 with 95% of value differences <3 mg/L. CONCLUSIONS: Selective measurement of HMW adiponectin by ELISA is feasible; however, individual differences among immunoassays must be considered. The evaluated ELISAs exhibit analytical characteristics that allow their use as equivalent for Western blot analysis in larger clinical and epidemiological groups.

3/7/96 (Item 1 from file: 154)
DIALOG(R)File 154:MEDLINE(R)

18408256 PMID: 18235151 Record Identifier: PMC2390947
Available on
03/01/09
Reduced albuminuria with sarpogrelate is accompanied by a
decrease in
monocyte chemoattractant protein-1 levels in type 2 diabetes.
Ogawa Susumu; Mori Takefumi; Nako Kazuhiro; Ishizuka
Tsuneo; Ito
Sadayoshi
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Clinical journal of the American Society of Nephrology - CJASN
(United
States) Mar 2008, 3 (2) p362-8, ISSN 1555-905X--Electronic
Journal Code: 101271570
Publishing Model Print-Electronic
Document type: Journal Article; Randomized Controlled Trial
Languages: ENGLISH
Main Citation Owner: NLM
Other Citation Owner: NLM
Record type: MEDLINE; Completed
BACKGROUND AND OBJECTIVES: Sarpogrelate has been shown to
reduce
albuminuria in diabetic nephropathy. For examination of whether
this is
based on the same mechanisms as angiotensin II receptor
blockers or
thiazolidinedione, effects of sarpogrelate on atherosclerotic
inflammatory
molecules and their relations to albuminuria in patients who had
diabetes
and had already been treated with angiotensin II receptor blockers
and with
or without thiazolidinedione were examined. DESIGN, SETTING,
PARTICIPANTS,
& MEASUREMENTS: Forty patients who had diabetes with
nephropathy and
arteriosclerosis obliterans and had already been treated with
angiotensin
II receptor blocker (n = 40) were randomly assigned to
sarpogrelate (300
mg/d; n = 20) or aspirin group (100 mg/d; n = 20). Plasma
monocyte

chemoattractant protein-1 and urinary albumin-to-creatinine ratio and
monocyte chemoattractant protein-1 were measured at baseline and 16 wk
after administration. RESULTS: Only the sarpogrelate group showed increases
in plasma adiponectin and decreases in both plasma and urinary
monocyte chemoattractant protein-1 and albumin-to-creatinine ratio levels.
Moreover, percentage change of monocyte chemoattractant protein-1 level
correlated positively to that of albumin-to-creatinine ratio. Even when the
sarpogrelate group was further divided into two groups with (n = 9) or
without thiazolidinedione (n = 11), changes in monocyte chemoattractant
protein-1 or albumin-to-creatinine ratio did not differ. CONCLUSIONS:
Sarpogrelate can reduce albuminuria and plasma and urinary monocyte
chemoattractant protein-1 levels while increasing plasma adiponectin
in diabetic nephropathy. These effects seem to be mediated via mechanisms
that are different from those of angiotensin II receptor blocker or
thiazolidinedione.
  Record Date Created: 20080229
  Record Date Completed: 20080623
  Date of Electronic Publication: 20080130

 3/7/97   (Item 2 from file: 154)
DIALOG(R)File 154:MEDLINE(R)

17390366   PMID: 16936907
   Adiponectin added into the plasma of healthy probands does not
affect platelet aggregability.
  Stejskal David; Proskova Jitka; Solichova Pavlina
  Department of Laboratory Medicine, Sternberk Hospital, Czech Republic.
david.stejskal@quick.cz
  Biomedical papers of the Medical Faculty of the University Palacky,
Olomouc, Czechoslovakia (Czech Republic) Jul 2006, 150 (1)
p89-90,
ISSN 1213-8118--Print Journal Code: 101140142
  Publishing Model Print
  Document type: In Vitro; Journal Article
  Languages: ENGLISH
  Main Citation Owner: NLM

Record type: MEDLINE; Completed
Six healthy non-obese probands without medical therapy and history of
disease were tested. In all of them platelet aggregability with addition of
human recombinant adiponectin in different concentrations (100; 75; 50 and 25 ng/l) were measured. It is concluded that increased level of
adiponectin has no significant antiaggregation effect on platelets from individuals without hypoadiponectinemia.
Record Date Created: 20060828
Record Date Completed: 20070823

3/7/98 (Item 3 from file: 154)
DIALOG(R)File 154:MEDLINE(R)

16729453 PMID: 16123486
Osteoprotegerin is associated with silent coronary artery disease in
high-risk but asymptomatic type 2 diabetic patients.
Avignon Antoine; Sultan Ariane; Piot Christophe; Elaerts Stephane;
Cristol Jean Paul; Dupuy Anne Marie
Metabolic Disease Department, Lapeyronie Hospital, 371, Av. Doyen G.
Giraud, 34295, Montpellier, Cedex 5, France.
a-avignon@chu-montpellier.fr
Diabetes care (United States) Sep 2005, 28 (9) p2176-80, ISSN
0149-5992--Print Journal Code: 7805975
Publishing Model Print
Document type: Journal Article
Languages: ENGLISH
Main Citation Owner: NLM
Record type: MEDLINE; Completed
OBJECTIVE: Osteoprotegerin (OPG) is an inhibitor of osteoclastogenesis,
which has been recently involved in atherosclerosis. The relationship
between coronary atherosclerosis and OPG has never been studied in
asymptomatic type 2 diabetic patients. RESEARCH DESIGN AND METHODS: This is
a nested case-control study; 162 asymptomatic type 2 diabetic patients were
evaluated for silent myocardial ischemia using stress myocardial perfusion
imaging; of 50 patients with positive results, 37 underwent coronary
angiography, 20 of whom showed significant coronary artery disease (CAD

group). Of 112 patients without silent myocardial ischemia, 20 subjects
(NO-CAD group) were selected and matched by age and sex to patients with
CAD. OPG, C-reactive protein, adiponectin , lipoprotein(a), albuminuria, and classical risk factors were measured. RESULTS: The
percentages of subjects with OPG levels above median and with nephropathy
were higher in the CAD group than in the NO-CAD group (70 vs. 25%, P =
0.004 and 50 vs. 5%, P = 0.001, respectively). LDL cholesterol levels were
higher and HDL cholesterol levels lower in the CAD compared with the NO-CAD
group (P = 0.033 and P = 0.005, respectively). No other variables were
associated with CAD. Logistic regression analysis showed that OPG values
above median (odds ratio 8.31 [95% CI 1.18-58.68], P = 0.034) and
nephropathy (21.98 [1.24-388.36], P = 0.035) were significant independent
predictors of asymptomatic CAD in type 2 diabetic patients. CONCLUSIONS:
Our investigation reports the first evidence of an independent association
of OPG with asymptomatic CAD in type 2 diabetic patients. The results of
this nested case- control study with 20 cases need to be confirmed in a
larger population.
  Record Date Created: 20050826
  Record Date Completed: 20051107

 3/7/99 (Item 4 from file: 154)
DIALOG(R)File 154:MEDLINE(R)

16277342 PMID: 15599338
   [Adiponectin gene polymorphism and protein dysfunction in the development of insulin resistance]
  Polimorfizm genu i zaburzenia funkcjonalne adiponektyny jako jedna z
przyczyn rozwoju opornosci na insuline.
  Karbowska Joanna; Warczak Elzbieta; Kochan Zdzislaw
  Katedra Biochemii Akademii Medycznej w Gdansku.
  Post py higieny i medycyny doswiadczalnej (Online) (Poland) 2004, 58
 p449-57, ISSN 1732-2693--Electronic Journal Code: 101206517
  Publishing Model Print

Document type: English Abstract; Journal Article; Review
Languages: POLISH
Main Citation Owner: NLM
Record type: MEDLINE; Completed
Adiponectin, an adipocyte-secreted protein encoded by the ACDC gene (also known as APM1), has been shown to play an important role in the regulation of fatty acid and glucose metabolism in liver and muscle, where it modulates insulin sensitivity. Adiponectin enhances fatty acid oxidation in liver and muscle, thus reducing triglyceride content in these tissues. Moreover, it stimulates glucose utilization in muscle and inhibits glucose production by the liver, consequently decreasing blood glucose levels. Plasma adiponectin levels are positively correlated with insulin sensitivity in humans. Circulating adiponectin forms a wide range of multimers. Mutations in the ACDC gene result in an impaired multimerization and/or impaired secretion of adiponectin from adipocytes, both linked to the development of insulin resistance and type II diabetes. This review focuses on the molecular mechanisms underlying hypoadiponectinemia associated with the diabetic phenotype. We further discuss the more recent findings that implicate adiponectin multimer formation as an important feature of the biological function of this adipocyte-derived hormone. (54 Refs.)
Record Date Created: 20041215
Record Date Completed: 20060421

3/7/100 (Item 1 from file: 399)
DIALOG(R)File 399:CA SEARCH(R)

150463748 CA: 150(22)463748k JOURNAL
The effects of pitavastatin, eicosapentaenoic acid and combined therapy
on platelet-derived microparticles and adiponectin in hyperlipidemic,
diabetic patients
AUTHOR(S): Nomura, Shosaku; Inami, Norihito; Shouzu, Akira; Omoto, Seitarou; Kimura, Yutaka; Takahashi, Nobuyuki; Tanaka, Atsushi; Urase, Fumiaki; Maeda, Yasuhiro; Ohtani, Hajime; Iwasaka, Toshiji
LOCATION: Division of Hematology, Kishiwada City Hospital, Kishiwada,
Japan,
JOURNAL: Platelets (Platelets) DATE: 2009 VOLUME: 20 NUMBER: 1
PAGES: 16-22 CODEN: PLTEEF ISSN: 0953-7104 LANGUAGE: English

PUBLISHER: Informa Healthcare
SECTION:
CA201008 Pharmacology
IDENTIFIERS: pitavastatin eicosapentaenoate combination platelet derived
microparticle hyperlipidemia diabetes antiatherosclerotic
DESCRIPTORS:
Antiarteriosclerotics...
antiatherosclerotics; pitavastatin, eicosapentaenoic acid and combined
therapy on platelet-derived microparticles and adiponectin
High-density lipoproteins...
HDL cholesterol, HDL-C; pitavastatin, eicosapentaenoic acid and
combined therapy on platelet-derived microparticles and adiponectin
Low-density lipoproteins...
LDL cholesterol, LDL-C; pitavastatin, eicosapentaenoic acid and
combined therapy on platelet-derived microparticles and adiponectin
Diabetes mellitus...
non-insulin-dependent; pitavastatin, eicosapentaenoic acid and combined
therapy on platelet-derived microparticles and adiponectin
Adiponectins... Atherosclerosis... CD40(antigen)... Combination chemotherapy... Diabetic angiopathy... Human... Hyperlipidemia... Platelet
aggregation inhibitors... Triglycerides...
pitavastatin, eicosapentaenoic acid and combined therapy on
platelet-derived microparticles and adiponectin
CAS REGISTRY NUMBERS:
57-88-5 biological studies, LDL and HDL cholesterol; pitavastatin,
eicosapentaenoic acid and combined therapy on platelet-derived
microparticles and adiponectin
10417-94-4 147511-69-1 pitavastatin, eicosapentaenoic acid and combined
therapy on platelet-derived microparticles and adiponectin

3/7/101 (Item 2 from file: 399)
DIALOG(R)File 399:CA SEARCH(R)

148423018 CA: 148(19)423018z JOURNAL
Relationship of multimers and genetic polymorphism of adiponectin with
type 2 diabetes
AUTHOR(S): Sun, Hong; Tan, Yuanming; Liu, Zhaoqian
LOCATION: Institute of Clinical Pharmacology, Central South University,
Changsha, Peop. Rep. China, 410078
JOURNAL: Guoji Bingli Kexue Yu Linchuang Zazhi (Guoji Bingli Kexue Yu

Linchuang Zazhi) DATE: 2006 VOLUME: 26 NUMBER: 5 PAGES: 432-435
  CODEN: GBKYAR ISSN: 1673-2588 LANGUAGE: Chinese PUBLISHER: Guoji
Bingli Kexue Yu Linchuang Zazhi Bianjibu
  SECTION:
    CA214000 Mammalian Pathological Biochemistry
  IDENTIFIERS: review adiponectin single nucleotide polymorphism
multimer
    type 2 diabetes
  DESCRIPTORS:
Diabetes mellitus...
    non-insulin-dependent; relationship of multimers and genetic
    polymorphism of adiponectin with type 2 diabetes
Adiponectins... Single nucleotide polymorphism...
    relationship of multimers and genetic polymorphism of adiponectin
with
    type 2 diabetes

 3/7/102   (Item 3 from file: 399)
DIALOG(R)File 399:CA SEARCH(R)

  146314021   CA: 146(16)314021h   JOURNAL
  Adiponectin inhibits osteoclast formation stimulated by
  lipopolysaccharide from Actinobacillus actinomycetemcomitans
  AUTHOR(S): Yamaguchi, Noboru; Kukita, Toshio; Li, Yin-Ji; Argueta,
Jose
Guillermo Martinez; Saito, Toshiyuki; Hanazawa, Shigemasa; Yamashita,
Yoshihisa
  LOCATION: Department of Preventive Dentistry, Kyushu University
Faculty
of Dental Science, Fukuoka, Japan,
  JOURNAL: FEMS Immunol. Med. Microbiol. (FEMS Immunology and Medical
Microbiology) DATE: 2007 VOLUME: 49 NUMBER: 1 PAGES: 28-34 CODEN:
FIMIEV ISSN: 0928-8244 LANGUAGE: English PUBLISHER: Blackwell
Publishing
Ltd.
  SECTION:
    CA214003 Mammalian Pathological Biochemistry
  IDENTIFIERS: adiponectin osteoclast lipopolysaccharide
Actinobacillus
    RANKL iNOS TLR4 NFkappaB, periodontal disease adiponectin
osteoclast
    lipopolysaccharide Actinobacillus RANKL
  DESCRIPTORS:
Adiponectins... Aggregatibacter actinomycetemcomitans...
Periodontium,disease... Osteoclast...
    adiponectin inhibited osteoclast formation stimulated by
    lipopolysaccharide from Actinobacillus actinomycetemcomitans via
RANKL,
    iNOS, TLR4, and NF-κB
Lipopolysaccharides...

bacterial; adiponectin inhibited osteoclast formation stimulated by
lipopolysaccharide from Actinobacillus actinomycetemcomitans via RANKL,
iNOS, TLR4, and NF-κB
Gene,animal...
iNOS; adiponectin inhibited osteoclast formation stimulated by
lipopolysaccharide from Actinobacillus actinomycetemcomitans via RANKL,
iNOS, TLR4, and NF-κB
Transcription factors...
NF-κB (nuclear factor of κ light chain gene enhancer in
B-cells); adiponectin inhibited osteoclast formation stimulated by
lipopolysaccharide from Actinobacillus actinomycetemcomitans via
Toll-like receptors...
TLR-4; adiponectin inhibited osteoclast formation stimulated by
lipopolysaccharide from Actinobacillus actinomycetemcomitans via RANKL,
iNOS, TLR4, and NF-κB
CAS REGISTRY NUMBERS:
501433-35-8 207621-35-0 adiponectin inhibited osteoclast formation
stimulated by lipopolysaccharide from Actinobacillus
actinomycetemcomitans via RANKL, iNOS, TLR4, and NF-κB
10102-43-9 biological studies, adiponectin inhibited osteoclast
formation
stimulated by lipopolysaccharide from Actinobacillus
actinomycetemcomitans via RANKL, iNOS, TLR4, and NF-κB

3/7/103 (Item 4 from file: 399)
DIALOG(R)File 399:CA SEARCH(R)

146050200 CA: 146(3)50200u PATENT
Pharmaceutical compositions of adiponectin variants and methods of storage
INVENTOR(AUTHOR): Zalevsky, Jonathan; Thi Nguyen, Duc-Hanh; Moore, Gregory L.; Ezhevsky, Sergei A.; Desjarlais, John R.; Chirino, Arthur J.;
Cash, Darian; Bernett, Matthew J.
LOCATION: USA
PATENT: U.S. Pat. Appl. Publ. ; US 20060276395 A1 DATE: 20061207
APPLICATION: US 2006421061 (20060530) *US 2005PV642476 (20050107) *US
2005PV650411 (20050203) *US 2005PV698358 (20050711) *US 2005PV720768 (20050926) *US 2005PV733137 (20051102) *US 2006328901 (20060109) *US 2006PV777825 (20060301) *US 2006PV781509 (20060309) *US 2006PV790220 (20060407)
PAGES: 79pp., Cont.-in-part of U.S. Ser. No. 328,901. CODEN: USXXCO
LANGUAGE: English
PATENT CLASSIFICATIONS:
CLASS: 514012000

IPCR/8 + Level Value Position Status Version Action Source Office:
A61K-0038/17 A I F B 20060101 20061207 H US
SECTION:
CA263005 Pharmaceuticals
CA201XXX Pharmacology
CA203XXX Biochemical Genetics
IDENTIFIERS: adiponectin variant pharmaceutical compn storage
DESCRIPTORS:
Human...
adiponectin variants; pharmaceutical compns. of adiponectin variants
and methods of storage
Drug delivery systems...
carriers; pharmaceutical compns. of adiponectin variants and methods of
storage
Stability...
improved; pharmaceutical compns. of adiponectin variants and methods of
storage
Solubility...
increased; pharmaceutical compns. of adiponectin variants and methods
of storage
Aggregation...
low, after storage; pharmaceutical compns. of adiponectin variants and
methods of storage
Protein sequences...
of adiponectin variants; pharmaceutical compns. of adiponectin variants
and methods of storage
Adiponectins... Storage... Molecular cloning... Protein engineering...
pharmaceutical compns. of adiponectin variants and methods of storage
Metabolic disorders...
treatment; pharmaceutical compns. of adiponectin variants and methods
of storage
Buffers...
10 mM PO4, 150 mM NaCl, storage in; pharmaceutical compns. of
adiponectin variants and methods of storage
CAS REGISTRY NUMBERS:
7647-14-5 biological studies, 10 mM PO4, 150 mM NaCl buffer, storage in;
pharmaceutical compns. of adiponectin variants and methods of storage
916465-32-2 916465-33-3 916465-34-4 916465-35-5 916465-36-6
916465-37-7 916465-38-8 unclaimed protein sequence; pharmaceutical
compns. of adiponectin variants and methods of storage
916465-15-1DP variants, amino acid sequence; pharmaceutical compns. of

adiponectin variants and methods of storage

3/7/104 (Item 5 from file: 399)
DIALOG(R)File 399:CA SEARCH(R)

145468690 CA: 145(24)468690s JOURNAL
High molecular weight adiponectin
AUTHOR(S): Horikoshi, Momoko
LOCATION: Grad. Sch. of Medicine, The Univ. of Tokyo, Tokyo, Japan,
JOURNAL: Igaku no Ayumi (Igaku no Ayumi) DATE: 2006 VOLUME: 217
NUMBER: 1 PAGES: 156-162 CODEN: IGAYAY ISSN: 0039-2359 LANGUAGE:
Japanese PUBLISHER: Ishiyaku Shuppan
SECTION:
CA214000 Mammalian Pathological Biochemistry
CA215XXX Immunochemistry
IDENTIFIERS: review adiponectin multimer diabetes insulin resistance
metabolic syndrome
DESCRIPTORS:
Cytokines...
adiponectin; relationship of high mol. wt. adiponectin with
insulin
resistance, diabetes and metabolic syndrome
Metabolic disorders...
metabolic syndrome X; relationship of high mol. wt. adiponectin
with
insulin resistance, diabetes and metabolic syndrome
Self-association...
multimerization; relationship of high mol. wt. adiponectin with
insulin
resistance, diabetes and metabolic syndrome
Diabetes mellitus... Human...
relationship of high mol. wt. adiponectin with insulin resistance,
diabetes and metabolic syndrome
CAS REGISTRY NUMBERS:
9004-10-8 biological studies, resistance; relationship of high mol.
wt.
adiponectin with insulin resistance, diabetes and metabolic
syndrome

3/7/105 (Item 6 from file: 399)
DIALOG(R)File 399:CA SEARCH(R)

145181041 CA: 145(10)181041v JOURNAL
Adiponectin - a key adipokine in the metabolic syndrome
AUTHOR(S): Whitehead, J. P.; Richards, A. A.; Hickman, I. J.;
Macdonald,
G. A.; Prins, J. B.
LOCATION: Centre for Diabetes and Endocrine Research, Princess
Alexandra

Hospital, University of Queensland, Brisbane, Australia
JOURNAL: Diabetes, Obes. Metab. (Diabetes, Obesity and Metabolism)
DATE: 2006 VOLUME: 8 NUMBER: 3 PAGES: 264-280 CODEN: DOMEF6
ISSN:
1462-8902 LANGUAGE: English PUBLISHER: Blackwell Publishing Ltd.
SECTION:
CA202000 Mammalian Hormones
IDENTIFIERS: review adiponectin insulin vascular inflammation
metabolic
syndrome
DESCRIPTORS:
Heart...
adiponectin secretion and circulating levels reduced in patient
with
coronary artery disease
Diabetes mellitus... Pancreas...
adiponectin secretion and circulating levels reduced in patient
with
diabetes
Human...
adiponectin secretion and circulating levels reduced in patient
with
diabetes and coronary artery disease
Cytokines...
adiponectin; thiazolidinedione suppress insulin resistance and
inflammation via adiponectin regulation in human
Peroxisome proliferator-activated receptors...
α; adiponectin receptor AdipoR2 was highly expressed in liver,
enhanced insulin sensitivity, reduced steatosis via activation of
AMPK
and increased peroxisome-proliferator-activated receptor-.al
Artery,disease...
coronary; adiponectin secretion and circulating levels reduced in
patient with coronary artery disease
Metabolic disorders...
metabolic syndrome X; adiponectin improved hepatic insulin
sensitivity,
increased fuel oxidn., decreased vascular inflammation, suggest
adiponectin replacement therapy can be used to treat metabolic s
Disease,animal...
steatosis; adiponectin receptor, AdipoR2 highly expressed in
liver,
enhanced insulin sensitivity, reduced steatosis via activation of
AMPK
and increased peroxisome-proliferator-activated receptor-.alp
Blood vessel,disease... Inflammation...
vasculitis; adiponectin decreased vascular inflammation in human
Cadherins...
13; T-cadherin expressed in endothelium, smooth muscle and
identified
as adiponectin-binding protein with preference for HMW adiponectin
multimer in human

CAS REGISTRY NUMBERS:
9004-10-8 biological studies, adiponectin improved hepatic insulin sensitivity in diabetic patient

3/7/106 (Item 7 from file: 399)
DIALOG(R)File 399:CA SEARCH(R)

144163988 CA: 144(10)163988j JOURNAL
Improvements in insulin resistance with weight loss, in contrast to rosiglitazone, are not associated with changes in plasma adiponectin or
adiponectin multimeric complexes
AUTHOR(S): Abbasi, Fahim; Chang, Sang-Ah; Chu, James W.; Ciaraldi, Theodore P.; Lamendola, Cindy; McLaughlin, Tracey; Reaven, Gerald M.; Reaven, Peter D.
LOCATION: Department of Medicine, Stanford University School of Medicine,
Stanford, CA, USA
JOURNAL: Am. J. Physiol. (American Journal of Physiology) DATE: 2006
VOLUME: 290 NUMBER: 1, Pt. 2 PAGES: R139-R144 CODEN: AJPHAP ISSN:
0002-9513 LANGUAGE: English PUBLISHER: American Physiological Society
SECTION:
CA201010 Pharmacology
CA202XXX Mammalian Hormones
IDENTIFIERS: thiazolidinedione insulin resistance wt loss adiponectin
multimer rosiglitazone
DESCRIPTORS:
Cytokines...
adiponectin, including multimeric complexes; improvements in insulin
resistance with wt. loss, in contrast to rosiglitazone, are not assocd.
with changes in plasma adiponectin or adiponectin multimeri
Human... Obesity...
improvements in insulin resistance with wt. loss, in contrast to
rosiglitazone, are not assocd. with changes in plasma adiponectin or
adiponectin multimeric complexes
Body weight...
loss; improvements in insulin resistance with wt. loss, in contrast to
rosiglitazone, are not assocd. with changes in plasma adiponectin or
adiponectin multimeric complexes
Diet...
restricted; improvements in insulin resistance with wt. loss, in

contrast to rosiglitazone, are not assocd. with changes in plasma adiponectin or adiponectin multimeric complexes
CAS REGISTRY NUMBERS:
50-99-7 biological studies, blood; improvements in insulin resistance with
wt. loss, in contrast to rosiglitazone, are not assocd. with changes in
plasma adiponectin or adiponectin multimeric complexes
9004-10-8 biological studies, improvements in insulin resistance with wt.
loss, in contrast to rosiglitazone, are not assocd. with changes in
plasma adiponectin or adiponectin multimeric complexes
122320-73-4 improvements in insulin resistance with wt. loss, in contrast
to rosiglitazone, are not assocd. with changes in plasma adiponectin or
adiponectin multimeric complexes

3/7/107 (Item 8 from file: 399)
DIALOG(R)File 399:CA SEARCH(R)

143432670 CA: 143(24)432670e PATENT
Materials and methods for modulating metabolism
INVENTOR(AUTHOR): Chan, Bill Piu; Wong, Gary Kwan Po; Xu, Jinxian; Chi,
Francis
LOCATION: Peop. Rep. China,
PATENT: U.S. Pat. Appl. Publ. ; US 20050245433 A1 DATE: 20051103
APPLICATION: US 2005118737 (20050429) *US 2004PV567899 (20040503) *US
2004PV637618 (20041220)
PAGES: 23 pp. CODEN: USXXCO LANGUAGE: English
PATENT CLASSIFICATIONS:
CLASS: 514003000; A61K-038/28A; A61K-031/4439B; A61K-031/426B;
A61K-031/13B
SECTION:
CA201010 Pharmacology
CA263XXX Pharmaceuticals
IDENTIFIERS: cysteamine compn hypercholesterolemia therapy diabetes
DESCRIPTORS:
Adipose tissue...
adipocyte, glut4 expression in; compns. and methods for treatment of
metabolic diseases
Cytokines...
adiponectin; compns. and methods for treatment of metabolic diseases
Adrenoceptor antagonists...
α-; compns. and methods for treatment of metabolic diseases

Heart,disease...
angina pectoris; compns. and methods for treatment of metabolic diseases
Adrenoceptor antagonists...
β-; compns. and methods for treatment of metabolic diseases
Hemorrhage...
cerebral; compns. and methods for treatment of metabolic diseases
Fatty acids,biological studies... Hypercholesterolemia... Anticholesteremic
agents... Hypolipemic agents... Obesity... Antiobesity agents...
Cardiovascular system,disease... Cardiovascular agents... Hypertension...
Antihypertensives... Hyperglycemia... Arteriosclerosis...
Antiarteriosclerotics... Antianginal agents... Prophylaxis... Ischemia...
Anti-ischemic agents... Combination chemotherapy... Fibrates... Platelet
aggregation inhibitors... Anticoagulants... Antidiabetic agents...
Sulfonylureas... Encapsulation... Proteins... Crown ethers...
Polyoxyalkylenes,biological studies... Polysiloxanes,biological studies...
Particle size... Bacilli... Human...
compns. and methods for treatment of metabolic diseases
Thrombosis...
coronary arterial; compns. and methods for treatment of metabolic diseases
Artery,disease...
coronary, thrombosis; compns. and methods for treatment of metabolic
diseases
Artery,disease...
coronary; compns. and methods for treatment of metabolic diseases
Metabolism,animal...
disorder, glucose intolerance; compns. and methods for treatment of
metabolic diseases
Transport proteins...
glucose transporter; compns. and methods for treatment of metabolic
diseases
Transport proteins...
GLUT-4 (glucose transporter 4); compns. and methods for treatment of
metabolic diseases
Liver... Muscle...
glut4 expression in; compns. and methods for treatment of metabolic
diseases
Drug delivery systems...
granules, enteric-coated; compns. and methods for treatment of
metabolic diseases
Drug delivery systems...

granules; compns. and methods for treatment of metabolic diseases
Brain,disease...
hemorrhage; compns. and methods for treatment of metabolic diseases
Lipoproteins...
high-d.; compns. and methods for treatment of metabolic diseases
Lipids,biological studies...
hyperlipidemia; compns. and methods for treatment of metabolic diseases
Glycerides,biological studies...
hypertriglyceridemia; compns. and methods for treatment of metabolic
diseases
Histamine receptors...
H2, blocker; compns. and methods for treatment of metabolic diseases
Autoimmune disease...
insulin-dependent diabetes mellitus; compns. and methods for treatment
of metabolic diseases
Diabetes mellitus...
insulin-dependent; compns. and methods for treatment of metabolic
diseases
Artery,disease...
intermittent claudication; compns. and methods for treatment of
metabolic diseases
Arm... Leg...
ischemia in; compns. and methods for treatment of metabolic diseases
Lipoproteins...
low-d.; compns. and methods for treatment of metabolic diseases
Albumins,biological studies...
microalbumin; compns. and methods for treatment of metabolic diseases
Diabetes mellitus...
non-insulin-dependent; compns. and methods for treatment of metabolic
diseases
Antidiabetic agents...
oral; compns. and methods for treatment of metabolic diseases
Drug interactions...
pharmacodynamic; compns. and methods for treatment of metabolic
diseases
Bile acids...
resins; compns. and methods for treatment of metabolic diseases
Drug delivery systems...
solids; compns. and methods for treatment of metabolic diseases
Brain,disease...
stroke; compns. and methods for treatment of metabolic diseases
Zeolites(synthetic),biological studies...
Zeolites (synthetic); compns. and methods for treatment of metabolic

diseases
CAS REGISTRY NUMBERS:
50-99-7 biological studies, blood, dysglycemia; compns. and methods for
treatment of metabolic diseases
69-93-2 biological studies, blood, hyperuricemia; compns. and methods for
treatment of metabolic diseases
59-67-6 9005-25-8 biological studies, compns. and methods for treatment
of metabolic diseases
9004-10-8 biological studies, hyperinsulinemia; compns. and methods for
treatment of metabolic diseases
12619-70-4D branched, compns. and methods for treatment of metabolic
diseases
60-23-1 61912-98-9 59112-80-0 156-57-0 75330-75-5 81093-37-0
79902-63-9 93957-54-1 134523-00-5 56211-40-6 50-78-2 56-03-1
2295-31-0 673-06-3 10238-21-8 29094-61-9 93479-97-1 64-77-7
94-20-2 135062-02-1 105816-04-4 56180-94-0 72432-03-2
122320-73-4
111025-46-8 11041-12-6 50925-79-6 182815-43-6 95522-45-5
52757-95-6 12619-70-4 9030-09-5 84337-62-2 107745-73-3
161973-57-5
79647-56-6 868350-96-3 657-24-9 compns. and methods for
treatment of
metabolic diseases
9004-34-6D 9004-54-0D 9005-25-8D derivs., compns. and methods for
treatment of metabolic diseases
9015-82-1 9001-42-7 inhibitor; compns. and methods for treatment of
metabolic diseases

3/7/108 (Item 9 from file: 399)
DIALOG(R)File 399:CA SEARCH(R)

142407211 CA: 142(22)407211c PATENT
Method of separating and assaying adiponectin multimer
INVENTOR(AUTHOR): Ebinuma, Hiroyuki; Yago, Hirokazu; Akimoto, Yuka;
Miyazaki, Osamu; Kadowaki, Takashi; Yamauchi, Toshimasa; Hara, Kazuo
LOCATION: Japan,
ASSIGNEE: Daiichi Pure Chemicals Co., Ltd.; Toudai Tlo, Ltd.
PATENT: PCT International ; WO 200538457 A1 DATE: 20050428
APPLICATION: WO 2004JP15260 (20041015) *JP 2003354930 (20031015)
PAGES: 40 pp. CODEN: PIXXD2 LANGUAGE: Japanese
PATENT CLASSIFICATIONS:
CLASS: G01N-033/53A; G01N-027/447B
DESIGNATED COUNTRIES: AE; AG; AL; AM; AT; AU; AZ; BA; BB; BG; BR;
BW; BY;
BZ; CA; CH; CN; CO; CR; CU; CZ; DE; DK; DM; DZ; EC; EE; EG; ES; FI;
GB; GD;

GE; GH; GM; HR; HU; ID; IL; IN; IS; JP; KE; KG; KP; KR; KZ; LC; LK;
LR; LS;
LT; LU; LV; MA; MD; MG; MK; MN; MW; MX; MZ; NA; NI; NO; NZ; OM; PG;
PH; PL;
PT; RO; RU; SC; SD; SE; SG; SK; SL; SY; TJ; TM; TN; TR; TT; TZ; UA;
UG; US;
UZ; VC; VN; YU; ZA; ZM; ZW DESIGNATED REGIONAL: BW; GH; GM; KE; LS;
MW; MZ
; NA; SD; SL; SZ; TZ; UG; ZM; ZW; AM; AZ; BY; KG; KZ; MD; RU; TJ; TM;
AT;
BE; BG; CH; CY; CZ; DE; DK; EE; ES; FI; FR; GB; GR; HU; IE; IT; LU;
MC; NL;
PL; PT; RO; SE; SI; SK; TR; BF; BJ; CF; CG; CI; CM; GA; GN; GQ; GW;
ML; MR;
NE; SN; TD; TG
  SECTION:
    CA209016 Biochemical Methods
    CA214XXX Mammalian Pathological Biochemistry
  IDENTIFIERS: adiponectin ELISA proteinase sepg multimer human blood
    disease diagnosis
  DESCRIPTORS:
Cytokines...
    adiponectin, including various multimers from human blood; method
of
    sepg. and assaying adiponectin multimer
Immunoassay...
    enzyme-linked immunosorbent assay; method of sepg. and assaying
    adiponectin multimer
Disease,animal...
    metabolic syndrome X; method of sepg. and assaying adiponectin
multimer
Disulfide group... Immunoassay... Human... Blood...
Albumins,reactions...
Kidney,disease... Liver,disease... Arteriosclerosis... Obesity... Gel
permeation chromatography... Gel electrophoresis...
Digestion,chemical...
Sample preparation...
    method of sepg. and assaying adiponectin multimer
Diabetes mellitus...
    non-insulin-dependent; method of sepg. and assaying adiponectin
    multimer
Antibodies and Immunoglobulins...
    to albumin, to adiponectin; method of sepg. and assaying
adiponectin
    multimer
  CAS REGISTRY NUMBERS:
9001-92-7 9003-05-8 9014-01-1 66676-43-5 209864-06-2 305344-27-8
    method of sepg. and assaying adiponectin multimer

 3/7/109   (Item 10 from file: 399)
DIALOG(R)File 399:CA SEARCH(R)

142322694 CA: 142(17)322694n PATENT
Adiponectin secretion enhancers containing plant extracts and/or their
microbial conversion products, and their use in antiarteriosclerotics,
antiobesity agents, antidiabetics, food additives, functional foods, and
feed additives
INVENTOR(AUTHOR): Akihisa, Toshihiro; Kobayashi, Masaki; Higashio, Chie;
Takahashi, Akira
LOCATION: Japan,
ASSIGNEE: Enkaku Iryou-Laboratories Co., Ltd.
PATENT: Japan Kokai Tokkyo Koho ; JP 200568132 A2 DATE: 20050317
APPLICATION: JP 2004143282 (20040513) *JP 2003287984 (20030806)
PAGES: 21 pp. CODEN: JKXXAF LANGUAGE: Japanese
PATENT CLASSIFICATIONS:
CLASS: A61K-035/78A; A23K-001/16B; A23L-001/30B; A61K-031/07B; A61K-031/575B; A61K-031/704B; A61P-003/04B; A61P-003/10B; A61P-009/10B;
C12Q-001/68B
SECTION:
CA263004 Pharmaceuticals
CA201XXX Pharmacology
CA211XXX Plant Biochemistry
CA217XXX Food and Feed Chemistry
CA218XXX Animal Nutrition
IDENTIFIERS: adiponectin secretion enhancer plant ext
antiarteriosclerotic, rice Momordica chrysanthemum adiponectin
secretion enhancer, rye Betula Alpinia adiponectin secretion enhancer,
shimeji ergosterol adiponectin secretion enhancer antiobesity,
antidiabetic adiponectin secretion enhancer plant ext, food feed
additive adiponectin secretion enhancer
DESCRIPTORS:
Sterols... Triterpenes... Antiarteriosclerotics... Antiobesity agents...
Antidiabetic agents... Food additives... Health food... Feed additives...
Blood serum... Drug bioavailability... Lyophyllum aggregatum...
Chrysanthemum... Secale cereale... Betula platyphylla japonica... Alpinia
zerumbet... Hypsizygus marmoreus...
adiponectin secretion enhancers contg. plant exts. and/or their
microbial conversion products for antiarteriosclerotics, antiobesity
agents, antidiabetics, food additives, functional foods, and feed a
Cytokines...
adiponectin, secretion enhancers; adiponectin secretion enhancers

contg. plant exts. and/or their microbial conversion products for antiarteriosclerotics, antiobesity agents, antidiabetics, food addit
Oryza sativa...
bran; adiponectin secretion enhancers contg. plant exts. and/or their
microbial conversion products for antiarteriosclerotics, antiobesity
agents, antidiabetics, food additives, functional foods, and Gene,animal...
expression; adiponectin secretion enhancers contg. plant exts. and/or
their microbial conversion products for antiarteriosclerotics, antiobesity agents, antidiabetics, food additives, functional foods
Momordica grosvenori...
fruit; adiponectin secretion enhancers contg. plant exts. and/or their
microbial conversion products for antiarteriosclerotics, antiobesity
agents, antidiabetics, food additives, functional foods, and Peroxisome proliferator-activated receptors...
γ, gene expression enhancement; adiponectin secretion enhancers contg. plant exts. and/or their microbial conversion products for antiarteriosclerotics, antiobesity agents, antidiabetics, food a
Bran...
rice; adiponectin secretion enhancers contg. plant exts. and/or their
microbial conversion products for antiarteriosclerotics, antiobesity
agents, antidiabetics, food additives, functional foods, and Arteriosclerosis... Obesity... Diabetes mellitus...
therapeutic agents; adiponectin secretion enhancers contg. plant exts.
and/or their microbial conversion products for antiarteriosclerotics,
antiobesity agents, antidiabetics, food additives, function
CAS REGISTRY NUMBERS:
848168-94-5 848168-95-6 848168-96-7 848168-97-8 848168-98-9
848168-99-0 848169-00-6 848169-01-7 848169-02-8 848169-03-9
adiponectin secretion enhancers contg. plant exts. and/or their
microbial conversion products, and their use in antiarteriosclerotics,
antiobesity agents, antidiabetics, food additives, functional foods,
and feed additives
469-38-5 57576-29-1 88901-36-4 57-87-4 35176-46-6 21238-33-5
106774-76-9 409349-92-4 adiponectin secretion enhancers contg. plant
exts. and/or their microbial conversion products for antiarteriosclerotics, antiobesity agents, antidiabetics, food additives, functional foods, and feed additives

```
 3/7/110      (Item 11 from file: 399)
DIALOG(R)File 399:CA SEARCH(R)
(c) 2009 American Chemical Society. All rts. reserv.

  142273179    CA: 142(15)273179n    JOURNAL
  Extrapancreatic effects of glimepiride - focusing on
antiarteriosclerotic
  action
  AUTHOR(S): Takayama, Hideichi
  LOCATION: Medical Affairs Department, Aventis Pharma Japan, Japan,
  JOURNAL: BIO Clin. (BIO Clinica)  DATE: 2004  VOLUME: 19  NUMBER: 13
  PAGES: 1100-1105  CODEN: BCILCY  ISSN: 0919-8237  LANGUAGE: Japanese
  PUBLISHER: Hokuryukan
  SECTION:
    CA201000 Pharmacology
  IDENTIFIERS: review glimepiride antiarteriosclerotic
arteriosclerosis
    insulin resistance platelet
  DESCRIPTORS:
Cytokines...
    adiponectin; extrapancreatic effects of glimepiride as
    antiarteriosclerotic agent
Endothelium...
    coronary arterial; extrapancreatic effects of glimepiride as
    antiarteriosclerotic agent
Artery...
    coronary, endothelium; extrapancreatic effects of glimepiride as
    antiarteriosclerotic agent
Antiarteriosclerotics... Arteriosclerosis... Platelet aggregation
inhibitors...
    extrapancreatic effects of glimepiride as antiarteriosclerotic
agent
Lipids,biological studies...
    metab.; extrapancreatic effects of glimepiride as
antiarteriosclerotic
    agent
  CAS REGISTRY NUMBERS:
10102-43-9  biological studies, extrapancreatic effects of
glimepiride as
    antiarteriosclerotic agent
9004-10-8  biological studies, resistance; extrapancreatic effects of
    glimepiride as antiarteriosclerotic agent
93479-97-1  extrapancreatic effects of glimepiride as
antiarteriosclerotic
    agent
? ds

Set      Items   Description
S1       49381   ADIPONECTIN
S2         464   S1 AND (MULTIMER OR AGGREGAT?)
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S3        110   RD S2  (unique items)
?

---Logging off of Dialog---


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       02jun09 07:15:05 User226352 Session D1140.3
             $3.36    0.543 DialUnits File5
              $122.00  50 Type(s) in Format  7
           $122.00  50 Types
   $125.36  Estimated cost File5
             $0.24    0.032 DialUnits File6
     $0.24  Estimated cost File6
             $0.54    0.084 DialUnits File24
                $5.40  2 Type(s) in Format  7
             $5.40  2 Types
     $5.94  Estimated cost File24
             $9.52    0.334 DialUnits File34
              $165.60  20 Type(s) in Format  7
          $165.60  20 Types
   $175.12  Estimated cost File34
             $0.17    0.023 DialUnits File40
     $0.17  Estimated cost File40
             $0.12    0.019 DialUnits File41
     $0.12  Estimated cost File41
             $0.55    0.107 DialUnits File45
                $8.64  4 Type(s) in Format  7
             $8.64  4 Types
     $9.19  Estimated cost File45
             $0.31    0.065 DialUnits File50
     $0.31  Estimated cost File50
             $0.22    0.051 DialUnits File65
                $1.30  1 Type(s) in Format  7
             $1.30  1 Types
     $1.52  Estimated cost File65
             $1.77    0.162 DialUnits File71
                $2.60  1 Type(s) in Format  7
             $2.60  1 Types
     $4.37  Estimated cost File71
             $3.54    0.255 DialUnits File72
               $61.28  16 Type(s) in Format  7
            $61.28  16 Types
    $64.82  Estimated cost File72
             $1.74    0.125 DialUnits File73
     $1.74  Estimated cost File73
             $0.18    0.028 DialUnits File76
     $0.18  Estimated cost File76
             $0.16    0.037 DialUnits File98
     $0.16  Estimated cost File98
             $0.27    0.042 DialUnits File103
     $0.27  Estimated cost File103
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          $0.12    0.019 DialUnits File136
     $0.12  Estimated cost File136
          $0.12    0.037 DialUnits File143
     $0.12  Estimated cost File143
          $0.78    0.153 DialUnits File144
            $1.92  1 Type(s) in Format  7
          $1.92  1 Types
     $2.70  Estimated cost File144
          $0.83    0.237 DialUnits File154
            $0.96  4 Type(s) in Format  7
          $0.96  4 Types
     $1.79  Estimated cost File154
          $0.39    0.111 DialUnits File155
     $0.39  Estimated cost File155
          $0.37    0.060 DialUnits File156
     $0.37  Estimated cost File156
          $0.26    0.056 DialUnits File162
     $0.26  Estimated cost File162
          $0.39    0.028 DialUnits File172
     $0.39  Estimated cost File172
          $0.27    0.019 DialUnits File305
     $0.27  Estimated cost File305
          $0.07    0.019 DialUnits File369
     $0.07  Estimated cost File369
          $0.05    0.014 DialUnits File370
     $0.05  Estimated cost File370
          $0.08    0.028 DialUnits File393
     $0.08  Estimated cost File393
          $4.00    0.306 DialUnits File399
            $32.78  11 Type(s) in Format  7
         $32.78  11 Types
    $36.78  Estimated cost File399
          $0.79    0.028 DialUnits File434
     $0.79  Estimated cost File434
          OneSearch, 29 files,  3.021 DialUnits FileOS
     $1.06  TELNET
   $434.75  Estimated cost this search
   $434.77  Estimated total session cost   3.413 DialUnits
Logoff: level 05.24.00 D  07:15:05
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